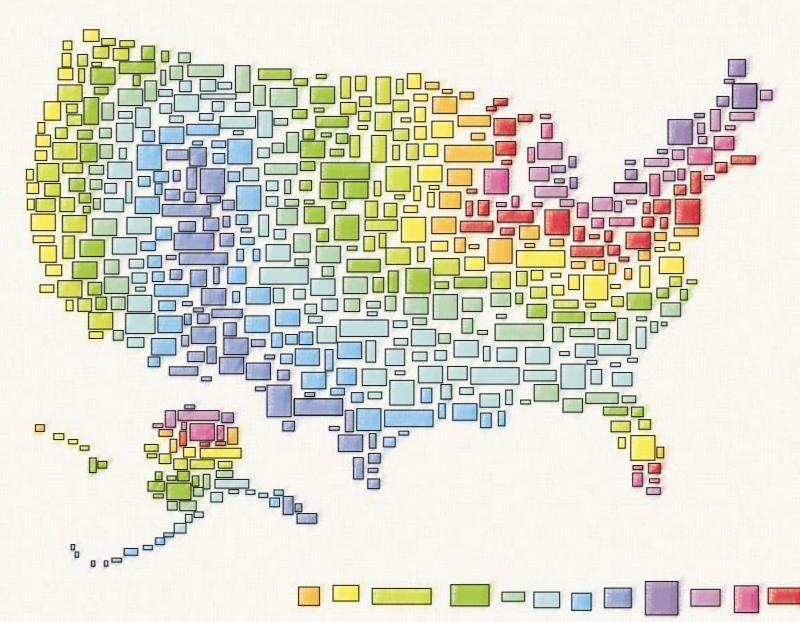
ATLAS OF UNITED STATES MORTALITY





FROM THE CENTERS FOR DISEASE CONTROL AND PREVENTION NATIONAL CENTER FOR HEALTH STATISTICS







ATLAS OF UNITED STATES MORTALITY

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MAPS

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Introduction

Maps have played a fundamental role in public health since the mid-1800's. Soon after a call for studying the geographic patterns of disease (1), Dr. John Snow linked the London cholera epidemic to a contaminated water supply (2). For over a hundred years afterward, however, the usefulness of mapping health outcomes in the United States was limited to either detailed views of a single area or national maps at a State or regional level. Then in 1975, when computer systems had become sufficiently powerful to automate the mapping process, the National Cancer Institute published maps of U.S. cancer death rates at the small-area level (3). Previously unnoticed clusters of high-rate counties on these maps led to numerous field studies, which uncovered, for example, the links between shipyard asbestos exposure and lung cancer (4) and snuff dipping and oral cancer (5). This first atlas demonstrated that mapping small-area death rates could be a valuable public health tool by generating etiologic hypotheses and identifying highrate communities where intervention efforts might be warranted. Its publication was followed by others from the National Cancer Institute (6-9) and instigated similar efforts around the world (10). Following the success of these atlases in advancing the understanding of cancer etiology, this monograph presents maps of the leading causes of death in the United States for the period 1988-92.

The research underlying this project has led to improved statistical methods for modeling death rates and innovative presentation formats for maps and graphics based on cognitive research (11). In this atlas, information previously available only in tabular form or summarized on a single map is presented on multiple maps and graphs. Broad geographic patterns by age group are highlighted by application of a new smoothing algorithm, and the geographic unit for mapping is defined on the basis of patterns of health care. These new features allow the public health researcher to examine the data at several geographic levels – to read an approximate rate for an area, to discern clusters of similar-rate areas, to visualize broad geographic patterns, and to compare regional rates. With these additional tools, important geographic patterns of cause-specific mortality in the United States can more easily be identified.

Although many causes of death included in this atlas have been mapped before, previous efforts have focused on a limited range of causes (3, 6–9, 12) or have presented data only at the State level (13). This is the first publication of maps of all leading causes of death in the United States on a small-area scale. Comparisons of map patterns across causes of death, sex, or race can provide clues to disease etiology. For this reason unlike many earlier atlases, separate maps by sex and race are included in the same volume, using consistent methods of presentation.

Data sources

The death rates mapped in this volume were calculated from information recorded on all U.S. death certificates to residents of the 50 States and the District of Columbia for 1988–92 and from population data for 1990.

Mortality. Numbers of deaths by age, race, sex, place of residence, and cause of death are based on original death certificates reported to the National Center for Health Statistics (NCHS) from the States. Death certificates with age not stated were excluded (0.025 percent of total). Race was classified following standard procedures for all United States vital statistics (14). Hispanic origin is classified separately from race. Hispanics are included in the data mapped in this atlas according to their race (white or black) as reported on the death certificate; but Hispanics with no racial designation are included in the "White" category. Deaths of persons of races classified as other than white or black were not mapped. Further details on the methods of data collection and processing of death certificates may be found in the Technical Appendix of Vital Statistics of the United States, 1990, Volume II, Mortality, Part A (14).

Population. Population counts from the 1990 Census (15), classified by age, race, sex, and county, were multiplied by 5 to create a denominator corresponding to the 5 years of mortality data. Rates computed using such a "person years at risk" denominator are often termed "average annual" rates. In the few instances where the calculated number of person years was less than the reported number of deaths, as when deaths occurred in a sparsely populated county before census enumeration, the person years at risk were inflated to equal the total number of deaths due to any cause.

Causes of Death

Mapped causes. The underlying causes of death were initially coded according to the *Ninth Revision, International Classification of Diseases* (ICD–9)(16) then aggregated according to the "List of 72 Selected Causes of Death," which is used in NCHS publications of tabular mortality statistics. This atlas includes maps of rates for the top ranking 11 causes of death from this 72-cause list (14), based on numbers of deaths in 1988–92, as well as the four leading cancer sites, motor vehicle injuries, and suicide and homicide by firearms—for a total of 18 causes of death. Specific definitions of the mapped causes of death are provided in table 1. Total

mortality rates due to any cause of death are also mapped. Unintentional injuries, homicides, and suicides are referred to as "external" causes of death. The other causes are referred to as "natural" causes. Death rates for a number of these causes are being used to monitor the health status of Americans at the State and national levels (17).

Quality of data. The issue of accuracy of cause-of-death statistics is fundamental to the interpretation of patterns shown on these maps (18–20). Because this accuracy has been challenged with regard to previously published mortality atlases, what follows is a discussion of potential sources of error in death certificate processing and reporting, and the means by which NCHS sought to compensate for these errors.

The quality of cause-of-death determination in the United States is affected by the accuracy and completeness of information—from medical diagnosis to final coding and processing of underlying cause of death. Beginning with mortality data for 1968, the underlying cause of death has been determined by an NCHS computerized system that consistently applies World Health Organization coding and selection rules to each death certificate using all conditions reported by the medical certifier (21). This system was last amended to incorporate the classification for Human immunodeficiency virus (HIV) infection, beginning with data year 1987. Automation of this task and crossverification of medical condition coding has reduced errors in assigning underlying cause from death certificate information to less than 1 percent (20). However, the completeness and accuracy of the information supplied on the certificate and the decedent's medical diagnosis remain as potential sources of error (22).

There are indications that the quality of medical information on the death certificate has improved over time. In particular, there has been a steady reduction of deaths assigned to the residual and nonspecific category of Symptoms, signs, and illdefined conditions (ICD-9 categories 780-799) from 1.5 percent before 1988 to 1.07 percent in 1992 (14). In addition, the number of medical conditions reported on death certificates has increased suggesting more detailed diagnostic information and greater care in completing the medical certification of death (23). Validation studies suggest that, for most broad categories, the reported underlying cause of death agrees well with hospital records of the decedents (24-26). However, for deaths that occur away from a medical setting—as for an unobserved sudden death—the medical certifier may not have

Table 1 - Causes of death for the NCHS mortality atlas: Definitions and map titles

Cause of death	Abbreviated map	ICD–9 category numbers - included	U.S. age-adjusted death rate, 1988–92¹			
	titles		White male	Black male	White female	Black female
Diseases of heart	Heart disease	390–398, 402, 404–429	205.0	282.9	104.8	171.7
Malignant neoplasms						
AII	All cancer	140-208	160.0	245.0	111.2	136.2
Trachea, bronchus, & lung	Lung cancer	162	56.1	84.5	25.7	26.0
Colon & rectum	Colorectal cancer	153-154, 159.0	16.6	21.1	11.2	15.4
Prostate	Prostate cancer	185	15.0	34.6	No data	No data
Female breast	Breast cancer	174	No data	No data	22.7	27.4
Cerebrovascular diseases	Stroke	430–438	28.1	56.6	23.8	43.4
Unintentional injuries						
All	Unintentional injuries	E800-E949	46.1	64.2	17.7	20.9
Motor vehicle injuries	Motor vehicle injuries	E810-E825	25.6	28.1	10.8	9.1
Chronic obstructive pulmonary diseases	COPD	490–496	27.5	26.4	15.5	11.0
Pneumonia & influenza	Pneumonia & influenza	480–487	17.2	27.9	10.3	13.5
Diabetes mellitus	Diabetes	250	11.1	23.7	9.4	24.9
Suicide						
All	Suicide	E950-E959	19.9	12.4	4.8	2.3
By firearms	Firearm suicide	E955.0-E955.4	12.9	7.8	2.0	1.0
Chronic liver disease & cirrhosis	Liver disease	571	11.7	19.5	4.9	8.4
Human immunodeficiency virus infection ²	HIV	*042–*044	14.7	46.9	1.1	10.3
Homicide & legal intervention						
All	Homicide	E960-E978	8.7	66.2	2.8	13.2
By firearms	Firearm homicide	E965.0-E965.4, E970	5.7	49.4	1.4	6.6
All causes	All causes	AII	648.5	1071.6	373.3	589.7

 $^{^{1}}$ Rate per 100,000 standard million population (See table 2.).

² Beginning with data for 1987, the National Center for Health Statistics introduced categories *042–*044 for classifying and coding human immunodeficiency virus infection. The asterisks before the categories are not part of the *Ninth Revision, International Classification of Diseases*.

sufficient information about the decedent's medical history to correctly report the underlying and contributing causes of death. For example, long-term diabetics are at high risk of heart disease and stroke as a consequence of their disease, but studies have shown substantial underreporting of diabetes on their death certificates (27). Other errors may occur where the cause of death is classified to a related, but incorrect, disorder or to a nonspecific disease category. The latter type of error can be addressed by grouping the related causes that are often confused or by not subsetting the broadly specified disease for analysis.

The potential for errors in assigning underlying cause of death was considered in defining the causes to map. Cause groups were created for this project that were broad enough to avoid these problems, yet specific enough to be meaningful for etiologic research. For example, cancers of the colon and rectum were combined because of the potential for misclassification between these diagnoses (25). All chronic obstructive pulmonary diseases (COPD) (including chronic bronchitis, emphysema, and asthma, ICD–9 categories 490–496) were combined for mapping because approximately 75 percent of all COPD deaths were coded as "Other," with the majority of these coded as "Not otherwise specified."

Amended data. The numbers of deaths that occurred in Alabama, Alaska, Hawaii, and New Jersey for the years 1988-92 are in error, because NCHS did not receive changes to the causes of death made at the State level (14, 28, 29). These differences are concentrated among selected causes of death, primarily the external causes. For example, the largest discrepancy found was for suicides in Alaska State records indicated 360 suicides during 1988-92, compared to 237 suicides reported to NCHS, a 34percent deficit. A comparison of annual death rates for 1979–92 due to unintentional injuries, motor vehicle injuries, suicides, and homicides in Alaska shows that rates for 1988-92 are in line with previous years except for suicides, which may be understated in this atlas. The reader is cautioned against overinterpretation of small-area rates for external causes in the previously mentioned States.

Incidence versus mortality. Although public health researchers would prefer to examine patterns of incidence rather than mortality, no nationwide registries exist for noncommunicable diseases. Though some problems surely remain in the mortality data presented here, the experience of National Cancer Institute epidemiologists in successfully following leads generated by cancer mortality

atlases demonstrates the utility of mapping these data (30).

GEOGRAPHIC UNIT

Deaths were initially assigned to a county (or equivalent administrative unit, such as independent city or parish) according to the residence of the deceased, regardless of the place of death. These 3,141 geographic units were then aggregated into Health Service Areas (HSA's) by a cluster analysis of where residents aged 65 years and over obtained routine short-term hospital care in 1988 (31) (appendix I). An HSA may be thought of as an area that is relatively self-contained with respect to hospital care. The original 802 HSA's defined by Makuc et al. (31) were supplemented to include Alaska and Hawaii. Also, several of the original HSA's were combined to achieve a minimum HSA size of 250 square miles for better visibility on the maps. Only New York City remains as a small but populous HSA; its HSA is enlarged for visibility east of its actual location and is labeled "NYC" on the maps. Several other major cities, such as Washington, D.C., were grouped with surrounding counties by the original cluster analysis. The final boundary file includes 805 HSA's.

HSA's are a compromise—in size and number—between the 3,141 counties and 50 States. Data for many of the causes included in this atlas are too sparse to provide stable 5-year rates at the county level, but mapping at the State level would mask many interesting geographic patterns in the data. (In fact, mapping at the HSA or county level may mask interesting local variations in the data. However, in addition to sparse data and confidentiality concerns, most States do not geocode death certificate addresses below the county level.)

Previously published cancer atlases (3, 6, 8, 9) mapped according to county or State Economic Area, aggregations of counties according to demographic and economic conditions in 1960. HSA's, defined on the basis of 1988 health care utilization, are more likely relevant for mapping current death rates and provide a reasonable spatial filter for detecting variations in death rates across the United States.

A map of the HSA boundaries is provided in appendix I, along with a listing of HSA names keyed to the boundary map by number. Each HSA name includes at least one county name and, in some cases, the name of a major city or town. These are provided for convenience in identifying HSA's on the maps. Each HSA that included counties from two

States (77 of 805 HSA's) was assigned to the State where the majority of its population lived. Further details are provided in appendix I.

For simplicity of presentation, boundary lines on the base map have been smoothed to within 5 miles of their original location (32). In addition, islands that were combined with a continental HSA to meet minimum size requirements are not shown; deaths among these island residents are included in the rates of the larger continental HSA. All maps were drawn using an Albers equal area projection (33).

This report examines the geographic effects of region as well as HSA. In this atlas, "region" is used in the generic sense and is not to be confused with

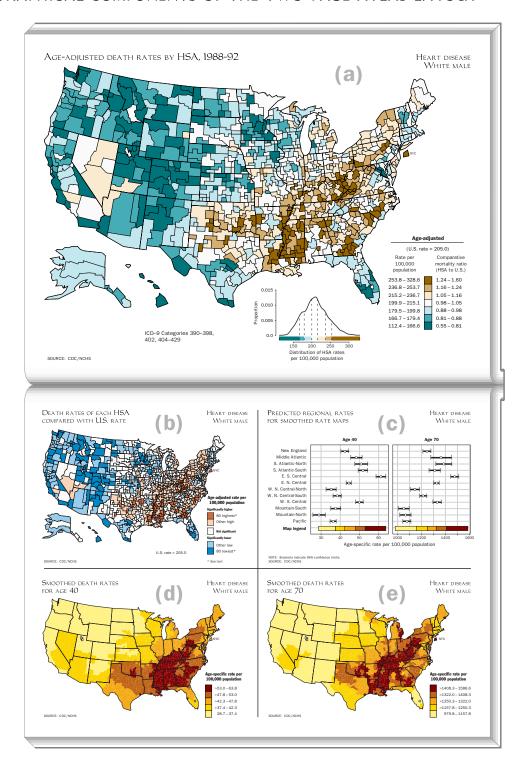
Census Regions (Northeast, Midwest, South, and West). Fourteen regions for whites and 12 regions for blacks were created by subdividing the nine Census Divisions (appendix I). For whites, the original South Atlantic, West North Central, and Mountain divisions were subdivided so that no region contained more than six States. Because of sparse populations, only the South Atlantic Division was subdivided for blacks. For whites and blacks, Alaska and Hawaii were considered as separate regions, apart from the remainder of the Pacific Division. Note that because an HSA can include counties from two States, the regional boundaries (appendix I) do not strictly follow State lines.

READER'S GUIDE

To aid the reader, the layout of graphical elements on each two-page set is fixed in terms of placement on the page, titles, and colors. It takes only a few minutes to become familiar with this

standard page layout (figure 1) and to read the "Graphical design" and "Statistical methods" sections, which explain the components below. The reader who does so will make full use of the integrated graphical presentation.

Figure 1. Graphical components of the two-page atlas layout



GRAPHICAL DESIGN

One of the unique features of this atlas is the use of cognitive research to guide its design. Early cognitive interviews and focus groups with typical atlas users at NCHS demonstrated a clear effect of a map's graphical design and page layout on the user's understanding of the underlying statistical information (34). Although any map could be used after some study, only easy-to-use maps encouraged repeated use and exploration of the data. To follow up on these early findings, the NCHS Office of Research and Methodology initiated a research effort by an interdisciplinary team of statisticians, psychologists, and geographers to examine how users cognitively process mapped information (11).

Before this research, few empirical studies had been conducted to evaluate the disparate map styles recommended in the literature, and none of these studies considered maps as complex as those in a national small-area mortality atlas. NCHS research revealed that the typical atlas audience of epidemiologists and other public health professionals wanted to (a) read an approximate HSA rate from a map, (b) identify clusters of areas with similar rates and regional patterns on the map, and (c) compare patterns across maps by cause, race, or sex. NCHS conducted collaborative and in-house experiments to examine the effects of basic map style, color scheme, pattern combination, and legend design on the ability of users to perform these specific tasks.

Basic map style. Experiments compared performance and preference using maps where rates were represented by area shading (choropleth), symbols, dot density, and color-coded lines (isopleth). These experiments showed the clear advantage of classed (categorized rate) choropleth maps over competing map styles. A symbol map is not a feasible design for hundreds of small areas, and the map audience was unsure how to interpret lines and dot density for aggregated data mapped to variable-sized geographic units (35, 36). Map style preference differed somewhat by professional discipline (37) although performance did not (38, 39).

Attempts to convey more specific information about the distribution of mapped rates through the use of unclassed maps (for example, where "darkness" of color is proportional to the actual rate) failed. Users also rejected proportional legend designs where the height of the legend box reflects the width of the rate interval. Map readers were either confused by the unfamiliar design (40) or were unable to distinguish among similar shades on the map (35).

Therefore, information about the rate distribution is separated from the legend in this atlas and shown instead as a density plot beneath the main map (figure 1a).

Color. Comparisons of color schemes confirmed cartographers' recommendations (33) that distinct hues or patterns facilitated reading a rate from the map but that a sequence of increasing "darkness" of a single hue associated with increasing rates facilitated identification of clusters or broad patterns (36, 41, 42). A double-ended scheme, where each of two hues represents rates above and below the median rate with levels of darkness increasing equally from the middle category to both extremes, can be used accurately for both tasks (43, 44). Therefore, a double-ended scheme was used for the age-adjusted maps (figures 1a, 1b), where identifying the value for a single HSA might be necessary, and a single-ended (monochromatic) scheme was used for the smoothed maps (figures 1d, 1e), where spatial pattern recognition is more important.

Final atlas map colors were chosen to avoid common color vision deficiencies and to balance levels of darkness (or lightness) so that no single color visually dominated a map (44). From a list of acceptable single and paired hues, colors for each of the three types of maps in this atlas were chosen so that no specific color appeared on more than one map and a hue was used consistently wherever it appeared (for example, reds for high rates and blues and greens for low rates).

Hatching. The addition of hatched lines over HSA's was found to accurately convey rate variance information to readers without hampering their ability to identify the underlying colors, and hence the patterns, on the maps (39, 45). Double-hatching with parallel white and black hatch lines allows visibility of the hatching over light and dark colors (figure 1a). Note that the map for white male heart disease (figure 1a) did not require hatching.

Regional rates. In several of the cognitive experiments, map users were asked to compare their estimates of average rates for several regions (35, 41). The variation of responses indicated that this was the most difficult of the questions posed to them. Therefore, to aid in evaluating broad spatial patterns, a rowplot (46) has been included to show confidence limits of model-based regional rates along with each map set (figure 1c).

Page layout. The final composite page layout for this atlas, with its combination of plots and several types of maps (figure 1), may initially seem complex. However, the variety of innovative presentation

formats for each set of rates accommodates multiple uses of the maps and different backgrounds of the users. For example, the pattern of age-adjusted rates can be seen on the full page map, and approximate rates can be determined for HSA's (figure 1a). The map in figure 1b indicates where rates are significantly different from the U.S. rate. The smoothed maps illustrate the broad patterns in age-specific rates (figures 1d, 1e), and the graphic (figure 1c) allows comparison of modeled regional effects.

STATISTICAL METHODS

The statistics mapped in this volume were computed by traditional methods and innovative statistical models. The new models permit examination of age-specific patterns, providing information that may be hidden by use of the traditional summary age-adjusted rate. All rates shown are death rates per 100,000 population. Age-adjusted rates were computed by the direct method (47) using the U.S. standard million population (table 2); these are mapped (figure 1a) and tested for significance compared to the U.S. rate (figure 1b).

Table 2. Standard million population used for age adjustment, proportional to total U.S. population in 1940

Age	Standard Population		
0–4 years	80,061		
5–14 years	170,355		
15–24 years	181,677		
25–34 years	162,066		
35–44 years	139,237		
45-54 years	117,811		
55–64 years	80,294		
65-74 years	48,426		
75–84 years	17,303		
85 years	2,770		
and over			
Total	1,000,000		

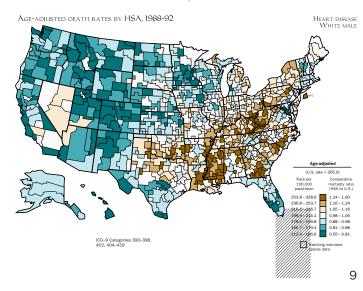
In addition, the age-specific numbers of deaths were modeled for each combination of race, sex.

cause, and place using mixed effects generalized linear models (48). Briefly, logarithms of the agespecific rates were modeled as a function of age, allowing each HSA to have a random intercept and, where possible, a random slope, within its particular region. Model results were used to compute improved variance estimates for the age-adjusted rates compared to traditional methods, to estimate regional effects, and to produce smoothed age-specific maps that reflected the broad spatial patterns in the data. Further details of this modeling effort are provided in appendix II.

Statistical methods used for each component of the two-page layout (figure 1) are discussed below.

Age-adjusted death rates by HSA, 1988–92. The age-adjusted rate map (figure 1a) presents the directly adjusted death rates for each HSA. An HSA has an overlaid hatch pattern if its rate has a coefficient of variation at least 23 percent. (The coefficient of variation is defined as the standard error of the rate divided by the rate, then multiplied by 100 and expressed as a percentage.) These rates have a large standard error because they are based on sparse data, typically fewer than 20 deaths, and therefore should be interpreted with caution. Note that the variance used for this calculation is the standard binomial variance estimator for directly age-adjusted rates (49) corrected by the model-based dispersion estimator. Refer to appendix II for details.

The rates are categorized according to percentiles of the rate distribution; the seven categories include, from minimum to maximum rate, respectively, 10 percent, 10 percent, 20 percent, 20 percent, 10 percent, and 10 percent of the 805 rates. These exact distributional percentiles were adjusted for mapping, if necessary, so that the legends accurately list the ranges of rates in each category, rounded to the number of digits shown. For example, a



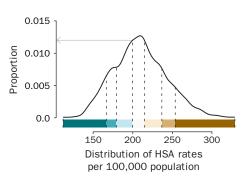
legend range of 5.2 to 10.3 includes all rates from 5.150 to 10.349.

In instances where over 10 percent of the 805 rates are zero (no deaths occurred), all HSA's with zero rates are assigned to the darkest green category. The percentage in the next category is reduced to reflect only nonzero rates up to the next percentile cutpoint. If more than 20 percent (or 40 percent) of the 805 rates are zero, all HSA's with zero rates are assigned to the lowest category as above; but the second (and third, if necessary) lowest category is labeled "No HSA" in the legend to indicate that this color category does not appear on the map. The exact percentage of zero rates is shown in a density plot for each age-adjusted rate map.

The legend also shows ranges for the comparative mortality ratio, defined as the HSA ageadjusted rate divided by the U.S. age-adjusted rate. For example, for an HSA rate range of 100 to 150 per 100,000 population and a U.S. rate of 100 per 100,000 population, the comparative mortality ratio range would be 1.00 (100/100) to 1.50 (150/100), indicating rates that are at least equal to, but no more than 50 percent greater than, the U.S. rate. Although the ratio ranges on the right side of the legend may appear to overlap, this is just the result of rounding after dividing the rate ranges by the constant U.S. rate.

Distribution of HSA death rates. The distribution of HSA death rates is shown graphically below the age-adjusted rate map. This density plot, which may be interpreted as a smoothed histogram, provides the proportion of the 805 rates with a particular value. The area under the curve sums to 1.0. In the example shown below, 1.2 percent (or 10) of the HSA rates are approximately equal to 200. The color bar below this graph shows the correspondence of the mapped rate categories (figure 1a) to the density plot (for example, the endpoints of

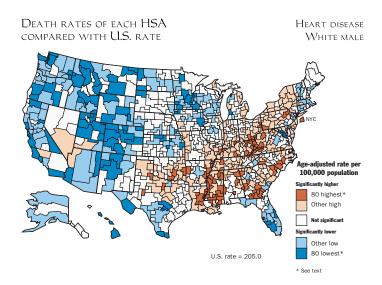
each segment of the color bar correspond to the cutpoints of the legend categories). For causes with extremely high outliers, the highest category on the



plot is truncated at the 99th percentile of the distribution, and the actual maximum rate is indicated above the rightmost color bar. This was done so that

the reader could see the shape of the distribution for every cause of death among blacks and for HIV among whites. For causes of death with no observed deaths in some HSA's, the proportion of zero rates is indicated by the height of a vertical line at zero (or an arrow to indicate that this proportion is beyond the scale of the graph).

Death rates of each HSA compared with the U.S. rate. This map (figure 1b) indicates whether each HSA rate is significantly different from the U.S. age-adjusted rate (α =0.05), which is shown below and to the left of the map legend. The significantly high rates are further subdivided into the highest 80 rates and other significantly high rates; significantly low rates are similarly subdivided. Note that the variance used for this hypothesis test is the standard binomial variance estimator for directly age-adjusted rates (49) corrected by the model-based dispersion estimator. Refer to appendix II for details.



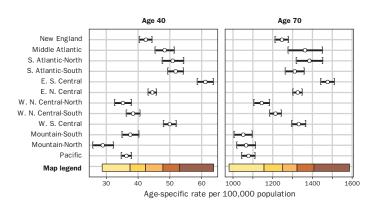
Smoothed rate maps and graphs. The remainder of the second page in each set (figures 1c, 1d, 1e) presents results from the statistical models (see appendix II). The geographic hierarchy included in the models provides estimates of the age-specific rates for each region and HSA. Separate graphs (figure 1c) or maps (figures 1d, 1e) are shown for two representative age groups. Ages 40 and 70 years are shown for natural causes of death, and ages 20 and 70 years are shown for external causes of death, which generally have higher rates for younger adults.

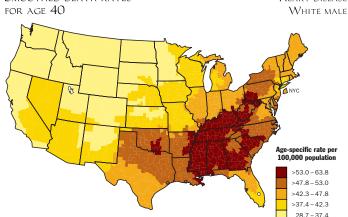
Predicted regional rates for smoothed rate maps. This plot provides the point estimates and 95percent confidence limits for the predicted agespecific regional rates. As was done for the ageadjusted rate map, a color bar is included reproducing











the legends of the corresponding smoothed agespecific maps (figures 1d, 1e). In instances where the maximum mapped HSA rate (figures 1d, 1e) is over four times the maximum predicted regional rate (figure 1c), this category bar is truncated on the graph and the actual maximum mapped HSA rate is shown inside the color bar.

Smoothed death rates for age 20, 40, or 70 years. These maps illustrate the broad spatial patterns in the age-specific death rates. HSA rates predicted by the models for the two representative ages (ages 40 and 70 years for natural causes and ages 20 and 70 years for external causes) were further smoothed using a two-dimensional weighted median smoothing algorithm and then categorized into quintiles of the rate distribution. Unlike the ageadjusted rate maps, these cutpoints were not adjusted to permit accurate reading of an HSA's rate, because the purpose of these maps is to show broad patterns. Instead, the legend ranges are shown as, for example, >12.2-14.0, where the upper limit of the next lower quintile is 12.2 (rounded to one decimal

place). Because the rates are color coded to show the relative ranking of the 805 HSA rates, the reader is cautioned to examine the legends carefully so as not to be misled by the usually great differences in the levels of rates for the younger and older age groups.

The original implementation of the smoothing algorithm (50) has been shown to retain important features of the data pattern better than competitive methods (51). The modification to include inverse standard error weights (52) gives more weight to rates based on large numbers of deaths, so that reliably estimated rates are less likely to be "smoothed out" of the map, even when they differ from rates in surrounding areas. Conversely, rates based on few deaths are more likely to be modified by the algorithm to appear similar to surrounding area rates. Thus, the smoothed map rates may not reflect the observed age-specific rate in a particular HSA. The reader is cautioned that although these maps accurately depict the expected level of age-specific rates in broad areas, they should not be used to estimate a rate for a single HSA.

As shown in tables 1 and 3 and figure 2, the leading causes of death are a mixture of natural and external causes. Heart disease and cancer are the leading causes of death for males and females, blacks and whites; but the relative order of other causes differs by race and, to a lesser extent, by sex. Clearly, heart disease and cancer exact a tremendous toll on public health, together causing over half of all deaths in the United States each year (table 3). However, homicide among black males and unintentional injuries among white and black males cause the greatest years of potential life lost before age 65 (53).

Figure 3 presents the age-specific death rates for the United States by sex and race for the causes mapped in this atlas. For most natural causes, death rates rise steadily with age, in some cases after an initial drop from higher rates in children under age 5. For external causes death rates peak in the age group 15–24 years and then either decline (homicide), level off (suicide), or rise again (unintentional injuries) in the age group 65 years and over. Greater differences in age-specific rates by sex and race are noted for the external causes of death. Exceptions to these general patterns are noted in the cause-specific discussions.

The age-adjusted rate maps (figure 1a) have been reproduced on a single page for each sex and race group (figure 4). These small maps have colors assigned according to the value of the comparative mortality ratio, that is, the proportional difference between each HSA's rate and the U.S. rate (more than

25 percent higher, 16 percent to 25 percent higher, within 15 percent of the U.S. rate, etc.). Through use of a common scale, geographic patterns of rates can be compared quickly. In addition, color coding according to the level instead of the relative ranking of the rates permits comparison of the range and variability of the rates across cause, race, and sex. Unlike the full-page maps (figure 1a), where rates are assigned to all seven color categories according to their ranks, using the absolute scaling of figure 4, all 805 rates could be coded to a single color if the range of rates is very narrow. For example, in figure 4 most HSA's have very low HIV death rates, with higher rates found in urban areas across the United States.

What follows are brief summaries about each cause of death along with comments on the geographic patterns of mortality. These notes are not intended to be comprehensive literature reviews. Information has been drawn heavily from annual statistical summaries published by NCHS, textbooks, and review articles. References are provided for additional information. Where appropriate, comments are provided on rates among Native Americans, Alaskan Natives, and Asian Americans for comparison to whites and blacks even though rates for other racial groups are not mapped in this atlas.

For each cause separate paragraphs include:

- General comments, including differences in national statistics over race, sex, and time;
- · generally accepted risk factors; and
- geographic patterns.

Table 3. Average annual number of deaths by cause, race, and sex during 1988–92

Cause of death	White male	Black male	White female	Black female	Total
Heart disease	323,842	37,866	323,103	38,825	723,636
All cancer	232,057	31,599	208,910	24,979	497,545
Lung cancer	78,790	10,455	45,023	4,474	138,742
Colorectal cancer	25,139	2,794	25,545	3,138	56,616
Prostate cancer	26,592	5,066	0	0	31,658
Breast cancer	0	0	37,928	4,623	42,551
Stroke	48,635	7,717	76,999	9,933	143,284
Unintentional injuries	51,020	8,970	25,673	3,732	89,395
Motor vehicle injuries	26,495	3,932	12,152	1,477	44,056
COPD	45,351	3,609	35,426	2,084	86,470
Pneumonia & influenza	31,554	4,025	37,430	3,329	76,338
Diabetes	16,506	3,059	21,390	4,982	45,937
Suicide	22,162	1,747	5,607	356	29,872
Firearm suicide	14,572	1,092	2,289	151	18,104
Liver disease	14,070	2,338	7,580	1,315	25,303
HIV	15,840	6,424	1,232	1,679	25,175
Homicide	8,862	9,573	3,034	2,163	23,632
Firearm homicide	5,948	7,274	1,493	1,054	15,769
All causes	955,814	145,886	909,128	121,149	2,131,977

Figure 2. Age-adjusted death rates by cause, race, and sex

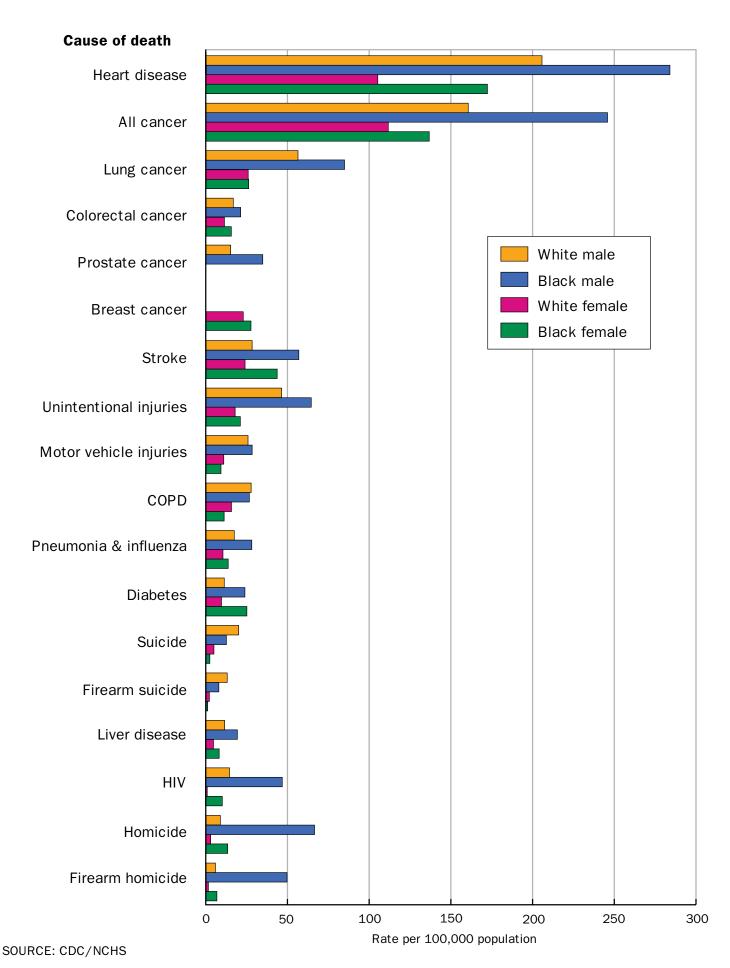
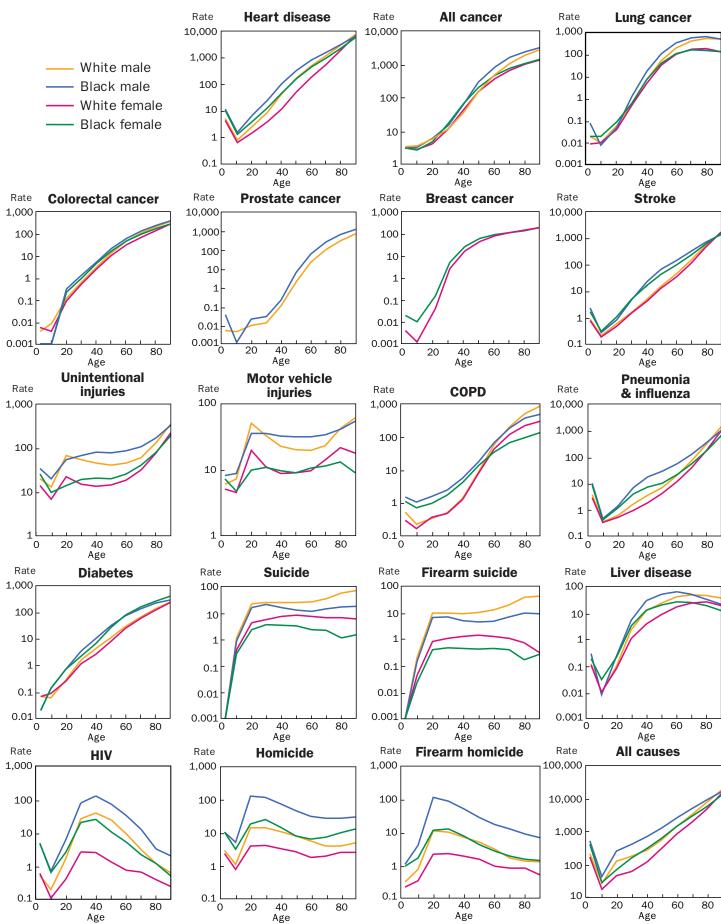
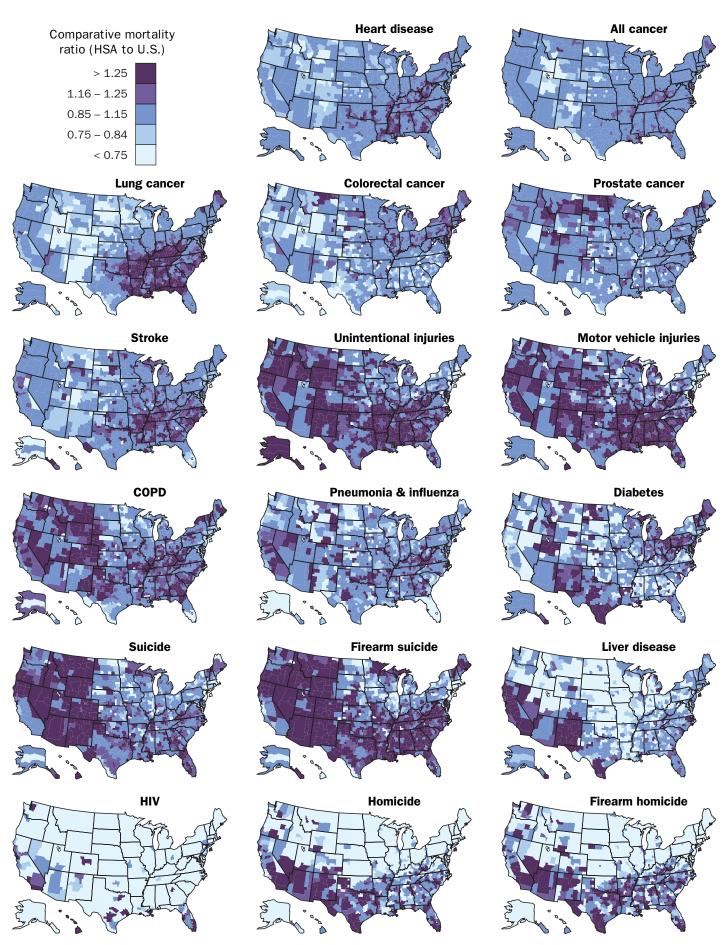
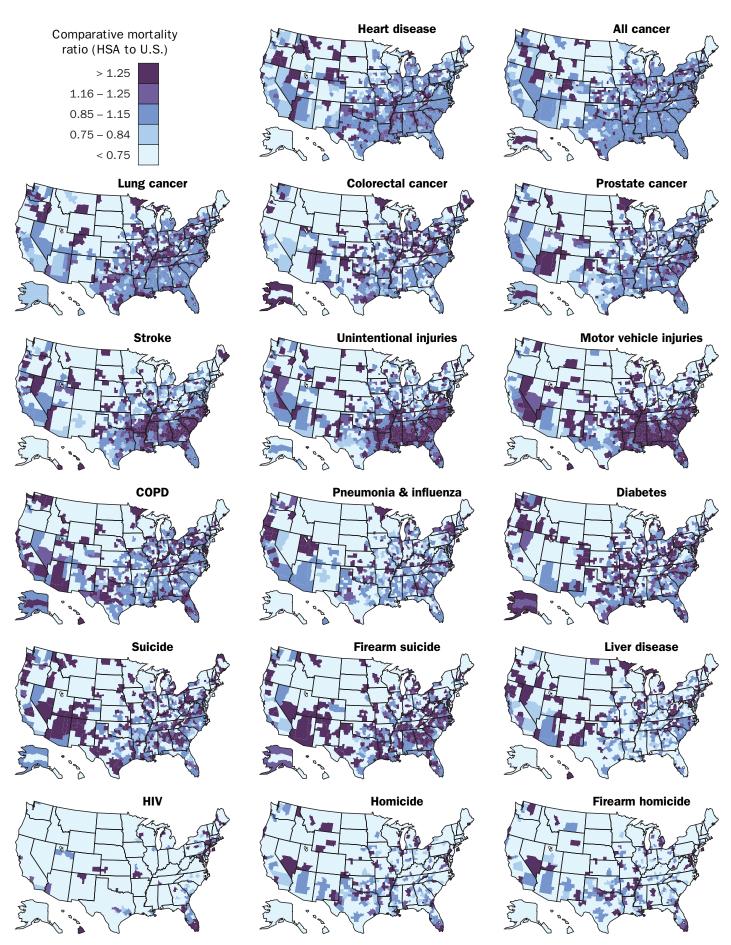


Figure 3. U.S. death rate per 100,000 population by age, cause, race, and sex

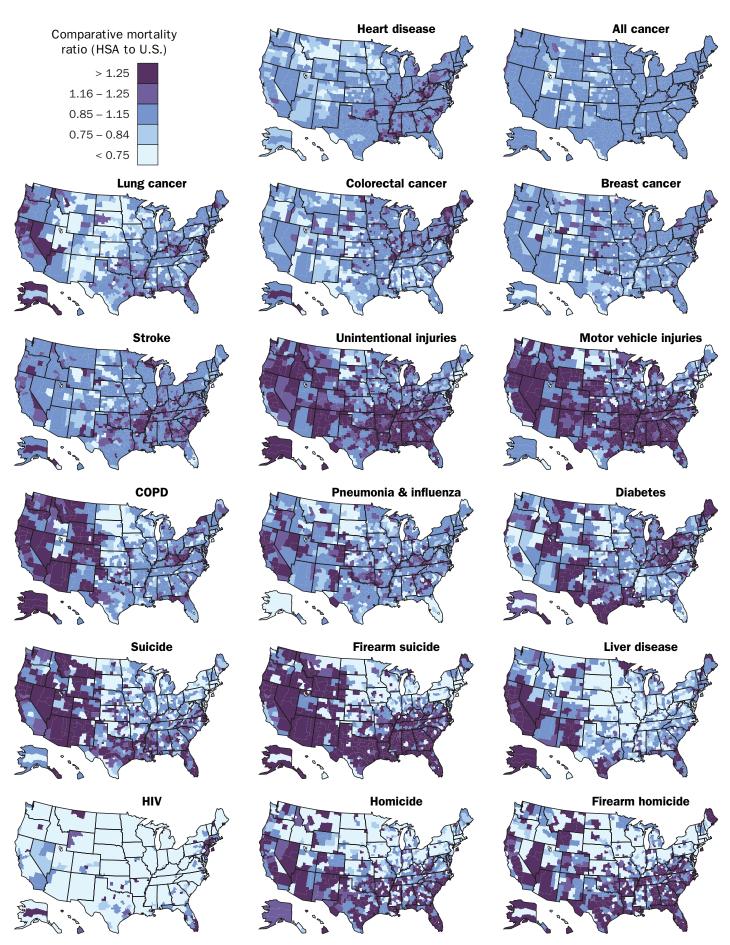


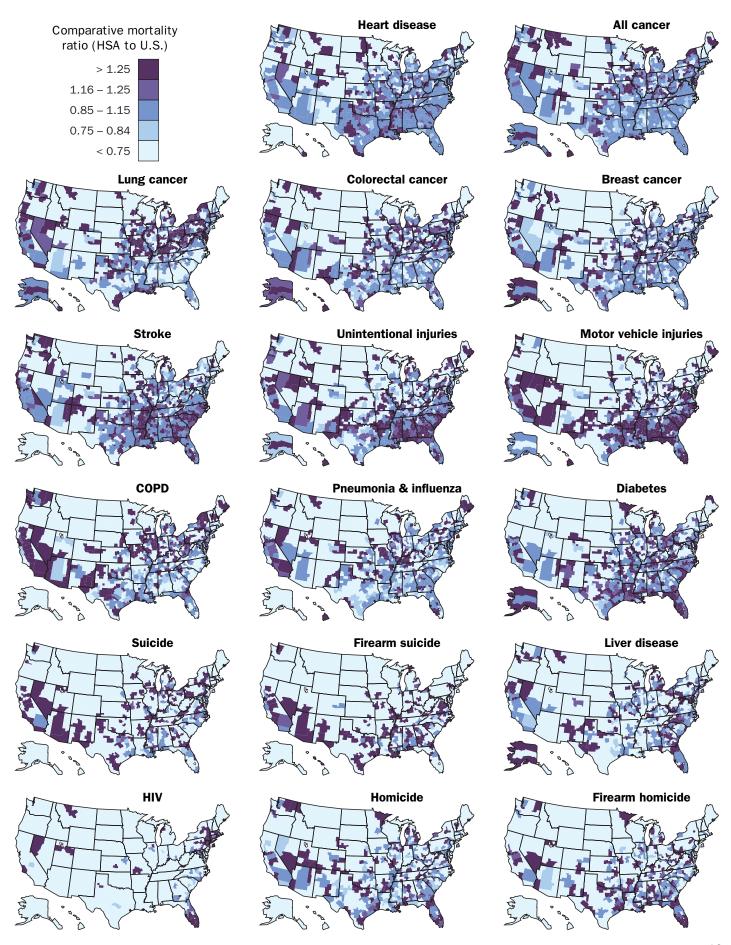
NOTE: For plotting purposes, rates equal to 0 are shown as 0.001 per 100,000 population. SOURCE: CDC/NCHS $\,$





SOURCE: CDC/NCHS 17





SOURCE: CDC/NCHS 19

HEART DISEASE

Although death rates have declined for over 30 years (54), heart disease remains the leading cause of death in the United States (53). Two-thirds of all deaths coded to this cause of death are specified as ischemic (or coronary) heart disease. Death rates from all diseases of the heart have been consistently higher for blacks than for whites for at least 40 years, with greater differences in recent years due to a more rapid drop in rates among whites (55). Hispanics, American Indians/Alaskan Natives, and Asians have lower rates than blacks or whites (53). Rates for women are 40 percent to 50 percent lower than the corresponding male rates in each racial or ethnic group. For men and women, regardless of race, death rates rise steadily with age, with lessening male/ female and black/white differences in the older age groups.

Along with age, sex, and race, other strong predictors of heart disease risk are cigarette smoking, high blood pressure, elevated serum cholesterol, physical inactivity, family history of heart disease, obesity, and diabetes (54, 56-59). Areas of current epidemiologic research include the effects of diet (60) and exercise (61) and the possible protective effect of estrogen therapy for postmenopausal women (62). The importance of these risk factors varies by age and sex and by the particular type of heart disease (63). Reductions in hospital discharges and hospitalized case fatality rates for heart disease patients point to improvements in population risk factor levels and medical care, for example, improved treatment of heart attack patients and wide availability of coronary artery bypass surgery, as partly responsible for continued reductions in death rates (64). However, over half of ischemic heart disease deaths among white males during a recent period occurred out of hospital or in emergency rooms; variation of this proportion by urbanization level and State suggests that a lack of quick access to effective emergency medical services is also a risk factor for death out of hospital (65).

In 1970 the highest heart disease death rates were in the Middle Atlantic region, whereas all States west of the Mississippi River had low rates (7). In the northeastern regions, rates were highest in metropolitan areas, but in the southeastern regions, nonmetropolitan areas had higher rates (66). Since 1970 rates first declined more rapidly in the northeastern United States than elsewhere, particularly in metropolitan areas, thus lessening regional differences (65, 67, 68). Later, rates declined

in nonmetropolitan areas and in southeastern States (66, 67). This geographic difference in mortality time trends resulted in a shift in the areas of highest rates from northeastern to southeastern States, although rates are still declining in all regions. This southeastern cluster of relatively high rates now includes areas west of the Mississippi River, where rates had been low in the past. A recent nationwide study showed a higher prevalence of self-reported current smoking in central cities across the United States for blacks and whites, with high rates among whites also noted in nonmetropolitan States in the South and West. The prevalence of high blood pressure and low education was also high in the nonmetropolitan South (64).

All cancer

Age-adjusted death rates for all cancer sites combined have changed little in the United States since the 1970's (53), while incidence rates have increased approximately 1.3 percent per year (69). However, these summary rates mask increases for specific types of cancer and decreases among younger persons that are attributed to improved diagnostic and therapeutic procedures (70). Over the past 20 years, overall incidence has increased at least 2 percent per year for cancer of the liver and kidney, as well as for melanoma of the skin and non-Hodgkin's lymphoma, prostate and testicular cancer among men, and lung cancer among women (69). Death rates for these cancer sites have also risen over this time, except for a 6-percent per year decline in testicular cancer (69). Reductions in incidence rates of over 2 percent per year during this period were seen for cancers of the uterus and cervix and in death rates for cancers of the stomach and uterine cervix and for Hodgkin's disease (69). Female lung cancer death rates have risen so rapidly that this is now the leading site of cancer mortality among women. (However, breast cancer incidence rates are more than double those of lung cancer (69), because of the high fatality rate among lung cancer patients.) Death rates from all cancers combined; cancers of the esophagus, uterine cervix, larynx, prostate, stomach and liver; and multiple myeloma are significantly higher in blacks (71); whereas whites experience higher rates of lymphomas; leukemias; and cancers of the ovary, brain, testis, and skin (69).

The most prominent risk factor for cancer development is cigarette smoking, which has been linked not only to lung cancer, but also to cancers of the mouth, pharynx, larynx, esophagus, pancreas,

uterine cervix, kidney, and bladder (71). Alcohol consumption in combination with smoking has been implicated in the development of cancers of the oral cavity, esophagus, and larynx (72). Studies of the influence of diet on cancer development have been inconclusive, though a diet high in fat and low in fiber, fruits, or vegetables has been associated with an increased risk for a number of cancers, including breast and colorectal cancer. Certain occupational exposures have been linked to leukemia, non-Hodgkin's lymphoma, lung cancer, bladder cancer, and liver cancer, and exposures to high levels of ionizing radiation are associated with cancers of the bone marrow, breast, and thyroid (72). The role of viruses, such as HIV and papilloma viruses, in the development of certain cancers is a topic of current research (72). Recent research has identified specific genetic abnormalities that may be responsible for a predisposition to breast and colorectal tumors (73, 74).

The geographic patterns for all cancer mortality reflect the patterns of death for leading cancer sites, particularly lung cancer. The four sites included in this atlas accounted for 54 percent of all cancer deaths during 1988–92.

LUNG CANCER

Lung cancer has been the leading cause of cancer death in men since the 1950's (71). In 1987 lung cancer became the leading cause of cancer death in women, surpassing breast cancer (69). Between 1950 and 1990, death rates increased 3.5 times among men and nearly 7 times among women (53), thus narrowing the gender gap in mortality for this tumor. During 1988–92, lung cancer death rates remained highest in black males, whose rates are 50 percent higher than white males; rates for black and white women are nearly equal.

The primary risk factor for lung cancer is cigarette smoking, an association demonstrated repeatedly in epidemiological studies (75). Risk has been shown to increase with the number of cigarettes smoked, the duration of smoking, an earlier age at onset of smoking, degree of inhalation, the tar and nicotine content, and the use of unfiltered cigarettes (75). Exposure to second-hand smoke; radon; and occupational exposures to asbestos, bis(chloromethyl) ether, polycyclic aromatic hydrocarbons, chromium, nickel, and inorganic arsenic compounds have also been shown to increase lung cancer risk (71, 75).

In the 1950's the highest lung cancer rates for white males were in northeastern urban areas and

port cities along the Atlantic and Gulf coasts (8). During the following 20 years, increasingly clustered areas of high rates appeared in the East South Central and South Atlantic-South regions, particularly along the Mississippi River (8). Case-control studies identified differences in smoking patterns and exposure to asbestos through shipyard employment as potential explanations for these geographic differences (8). From 1950 to 1970, patterns of lung cancer rates for white females were similar to earlier white male patterns, but a cluster of high rates appeared in Pacific States during the 1970's (8). During this time high rates for blacks were limited to urban centers in the Middle Atlantic and East North Central regions (9). The new maps for 1988–92 show patterns of even stronger geographic concentration among whites, with high male rates now extending along the Ohio River into the South Atlantic-North and East North Central regions. Rates for black males are highest in the South Atlantic and East South Central regions. The western cluster of high rates among white females now encompasses the entire Pacific region and portions of the Mountain regions; this western excess is most pronounced for the older ages, whereas higher rates in the East South Central, South Atlantic-South and New England regions are more notable for the younger age group.

Colorectal Cancer

From 1973 to 1992, incidence and mortality rates for colorectal cancer have increased by approximately 1 percent per year among blacks, while rates among whites have declined slightly (69). Improved survival rates are attributed to earlier detection due to improvements in diagnostic tests (76). From 1980 to 1992, black males experienced the highest death rates from colorectal cancer (53), though the incidence rates for white and black males are similar (76). Women are at a slightly lower risk of developing colorectal cancer (76).

In addition to age, race and sex, family or personal history of cancer or colorectal polyps, and inflammatory bowel disease are risk factors for colorectal cancer (71). Recent advances in identifying cancer susceptibility genes suggest that 10 percent of colorectal cancer cases are due to an inherited predisposition (73, 74). A number of studies have demonstrated that a high-fat, low-fiber diet can lead to the development of colorectal cancer, which could explain the 20- to 50-percent lower risk among Mormons and Seventh-Day Adventists, groups which observe certain dietary restrictions and refrain from

using alcohol and tobacco products (77). With increasing availability of methods of screening for colorectal cancer in asymptomatic patients (71), access to and utilization of these medical procedures may play a role in mortality patterns in the future.

Since the 1950's colorectal cancer death rates have been highest for blacks and whites in the more densely populated areas of the northeastern States, although regional differences have diminished over time (8, 9). For 1988–92 age-adjusted rates remain higher in the northeast (New England, Middle Atlantic, East North Central, and South Atlantic-North regions) but the age-specific maps show relatively higher rates among younger whites in South Central and South Atlantic States, although rates in this age group vary little across the United States.

Prostate Cancer

Prostate cancer death rates have increased slowly from 14.4 deaths per 100,000 population in 1980 to 16.6 deaths per 100,000 population during 1992 (53). Incidence rates have increased more rapidly, rising 50 percent over this period (71). This rapid rise in incidence is attributed to improved diagnostic techniques that may be identifying tumors that will never become life threatening (71). Black American men have the highest prostate cancer incidence, and possibly mortality, in the world (71). Their death rates are twice those of white American men (78). Prostate cancer is primarily a disease of older men, with over 80 percent of all diagnoses occurring over age 65 (71). Autopsy studies have estimated that 30 percent of men over age 50 have latent carcinoma of the prostate (79).

Very little is known about the etiology of prostate cancer, although age and race are recognized as major risk factors (71, 79). Other proposed risk factors include high circulating androgen levels (80), genetic predisposition to the disease (78), and dietary factors such as vitamin D deficiency (80) and high fat and vitamin A intake (79). Increased risk may be associated with exposure to cadmium and employment in agricultural, nitrate fertilizer, or ferrochromium industries (79). Other studies have suggested that a history of venereal disease or multiple sex partners also increases the risk of prostate cancer (78).

Scattered areas of high rates for white males have been noted previously in rural sections of the northern States (8). In the 1970's an apparent clustering of high black male death rates emerged in the Carolinas and central Florida (9). The 1988–92

maps for the older ages confirm these patterns for white and black men. Higher rates among younger men are seen along the Atlantic coast, but rates for this age group are extremely low.

Breast Cancer

In the United States, death rates for breast cancer have been stable for the past 50 years, even as incidence rates have increased (71). Before 1980 white women had slightly higher age-adjusted death rates than black women, but since then death rates for black women have increased 16 percent, surpassing rates for white women, which decreased 5 percent over the same period (53). Breast cancer incidence and death rates increase with age, but the rate of increase slows after menopause (81). Among premenopausal women, breast cancer incidence is higher in blacks (81).

Two well-established risk factors for breast cancer are personal history of breast, endometrial, or ovarian cancer and family history of breast cancer (82). The recent identification of genetic mutations linked to early onset breast cancer may explain at least part of this familial aggregation (73). Older age, higher socioeconomic status, and never having been married have been associated with increased risk of breast cancer (82). Other established risk factors include exposure to high doses of ionizing radiation; certain breast tissue abnormalities; and factors related to reproductive history such as early onset of first menarche, late onset of menopause, late age of first full-term pregnancy or nulliparity (83). The effects of alcohol consumption, oral contraceptive use, postmenopausal estrogen therapy, diet, and pesticide exposure are still being considered (82–84). As many as 70 percent of women diagnosed with breast cancer may not have any identifiable risk factor (83).

Between 1950 and 1980, the highest breast cancer death rates for white and black women were found in the urban areas of the New England and Middle Atlantic regions, although the north-south differences diminished over this period (8, 9). High rates for postmenopausal white females were concentrated in the urban areas of these northeastern States, but much of this excess can be explained by regional differences of recognized risk factors (85); premenopausal rates varied little by region (8). A comparison of the age-specific and age-adjusted maps for 1988–92 shows that the higher age-adjusted rates in the northeast are still predominantly due to geographic differences among older white women. The map for younger ages shows an east-to-west decline

in rates, but the range of rates is very narrow. High rates among black females appear to be scattered across the southern States for both age groups.

STROKE

In the United States, the death rate for cerebrovascular diseases (stroke) has steadily declined since about 1900 (86). Even so, stroke remains a leading cause of death, ranking third behind heart disease and all cancer for white and black women, fourth for white men, and fifth for black men. About 75 percent of strokes are ischemic (a blockage of cerebral blood vessels) rather than hemorrhagic (a ruptured blood vessel) (54). Sharp declines in ischemic stroke death rates are responsible for the downward trend in overall rates (56), although over half of the stroke deaths during 1988-92 were not coded to a specific type. Death rates for men have been higher than those for women since the 1950's, though the gender gap narrowed in the 1980's (86). During 1988-92 the age-adjusted death rate for stroke among blacks was twice that among whites for males and females, with the greatest racial disparities seen in the younger age groups (87). Rates among non-Hispanic whites and Asians were nearly equal during 1988-92, with rates among Hispanics and American Indians/Alaskan Natives somewhat lower (53). Hospitalization rates for stroke have remained stable over the period of declining mortality, although regional differences in these rates reflect those of mortality (87), suggesting that improvements in diagnosis and in prevention and treatment of associated medical conditions may have contributed to the reduction in mortality.

Risk factors for stroke vary somewhat according to the type of stroke in question (88), but one-third of all strokes may be attributed to a medical history of hypertension, diabetes, elevated hemoglobin levels, or prior heart disease (56). Other important risk factors for ischemic stroke include age, sex, race, genetic predisposition, cigarette smoking, obesity, elevated blood cholesterol and lipids, excess alcohol consumption, use of oral contraceptives, a history of transient ischemic attacks, and physical inactivity (54, 89, 90). At one time characteristics of soil and drinking water were suspected as risk factors for stroke, but the changing geographic patterns of stroke mortality are inconsistent with these hypotheses (91). Geographic and temporal differences in stroke mortality are now thought to be real, not an artifact of certification differences (92), and due to multiple individual, rather than community, factors (91).

Over 40 years ago, a cluster of areas with high stroke mortality was identified in the South Atlantic-South region, which became known as the "Stroke Belt" (91). Over time rates have declined faster in this region than others, lessening regional differences (92). Although rates remain high along the south Atlantic coast, the "Stroke Belt" appears to be dispersing as a consequence of regional differences in time trends, with clusters of relatively high rate areas seen more to the north and west of the original cluster (7, 91, 93). For example, the maps for 1988– 92 show that age-adjusted rates in the East South Central and West South Central regions are relatively high for whites and blacks. If the geographic differences noted in the smoothed maps by age group represent a cohort effect, high rates will continue to move westward. Little information is available about geographic patterns of risk factors for stroke, but a recent nationwide study showed self-reported hypertension and smoking to be high in the nonmetropolitan south (South Atlantic and South Central regions) (64). It is interesting to note some local exceptions to these regional trends, notably in retirement destinations. A study of stroke mortality by State of birth and death suggests that persons who retire to other regions retain the stroke risk of their States of origin; hence white retirees from the Middle Atlantic region may lower the total stroke death rate in Florida, compared to higher rates seen among white and black native Floridians (94).

Unintentional injuries

Death rates due to unintentional injuries have been decreasing among both sexes and all races since 1970 (53). Death rates from all unintentional injuries peak at ages 15–24 years, then rise again after age 60; in fact, unintentional injury was the leading cause of death for each age group from 1–4 years to 25–44 years in 1992 (53). For 1988–92 nearly half of these deaths were motor vehicle-related, but falls were the leading cause of unintentional injury death among older persons. Death rates among males were nearly three times those of females. Native Americans have the highest overall death rates due to unintentional injuries, and rates among blacks are somewhat higher than those among whites (53).

Age is the predominant risk factor for death due to unintentional injury. The very young, particularly males, are prone to unintentional injuries because their lack of knowledge and experience leads them to engage in high-risk behavior (95). The risk of sustaining a fatal injury increases with exposure to

potentially fatal materials, such as poisonous substances, or events, such as a dangerous work environment. The use of alcohol is a risk factor for many types of unintentional injuries such as drownings and motor vehicle crashes (96). Elevated death rates among older persons are attributed to their diminishing physical capacity, which leads to a higher frequency of accidents, a higher likelihood of sustaining an injury in the accident, and greater difficulty recovering from the injury (95).

The highest unintentional injury death rates are seen in the East South Central region for whites and the South Atlantic-South region for blacks, with scattered high-rate areas in the western States. Rates are higher in rural than in urban areas (12, 95). See cautionary note in "Amended data" section under "Causes of death."

Motor vehicle injuries

Rates of death due to motor vehicle injuries have declined to 42 percent of the 1970 U.S. rate (53). This improvement has been attributed to successful public awareness and education campaigns, legal interventions, improved vehicle and equipment designs, roadways, and emergency medical and trauma care (97). Despite these improvements, motor vehicle injury remains one of the leading causes of death among young adults. Motor vehicle death rates are nearly three times higher among males than among females (53). Native Americans have death rates about twice those for whites or blacks (97).

Factors that have been associated with motor vehicle fatalities include alcohol and drug use, risk denial and aggressive behavior, young or old age, and characteristics of the vehicle and roadways (95).

Motor vehicle-related death rates have been higher in the southeastern States and in scattered western areas (12, 95, 97). Motor vehicle death rates are higher in nonmetropolitan than metropolitan areas. The age-specific maps for 1988–92 show consistently high rates in the East South Central and South Atlantic-South regions, especially among young adults, but the location of high-rate areas in the west vary by race and sex. See cautionary note in "Amended data" section under "Causes of death."

CHRONIC OBSTRUCTIVE PULMONARY DISEASES

U.S. age-adjusted death rates for chronic obstructive pulmonary diseases (COPD) increased

from 1950 until 1988, after which rates remained relatively stable (53). Between 1980 and 1992, death rates increased among older age groups, while among the younger age cohorts death rates remained stable or declined (53). Since 1980 the largest increases in death rates for COPD have been among females (+76 percent) and among black men (+19 percent), but the highest rates are still seen among white males (53). Overall, male death rates are about twice those of females (53). This broad cause of death includes emphysema, asthma, and bronchitis, but less than 30 percent of deaths attributed to COPD during 1988–92 were coded to these specific causes.

Cigarette smoking and coal dust exposure are established risk factors for COPD (98). Other occupational dust and fume exposures, childhood lung disease, passive cigarette smoke exposure, prenatal cigarette smoke exposure (99), and low socioeconomic status as related to poor housing (98) are suggested risk factors. Familial aggregation of COPD has been noted, but it is not clear whether this is due to genetic predisposition or to shared environmental factors, such as dust or passive smoking (99).

Age-adjusted death rates are highest in the Mountain regions for whites and the South Atlantic regions for blacks. Rates are also high in other areas depending on race, sex, and age; for example, rates among older white males are high in the East South Central region, similar to patterns for lung cancer death rates. Patterns on these maps resemble those seen on earlier maps of emphysema (7), although care must be taken in drawing inferences about COPD component diseases.

PNEUMONIA & INFLUENZA

By 1980 the combined death rate for pneumonia and influenza declined to less than half the 1950 death rate (53); since then, the rates have changed little. A higher than usual proportion of deaths due to pneumonia and influenza occurred during the initial 4 years covered by this atlas (100, 101). Though influenza infection rates are highest among children and adolescents, nearly 90 percent of the deaths occur among older persons (53, 102). Death rates from pneumonia and influenza are 69 percent higher for males than for females and 48 percent higher for blacks than for whites. Pneumonia and influenza deaths have a characteristic annual pattern of peak death rates in the winter months and low death rates in the summer months, but additional deaths may occur during an epidemic (103).

The majority of risk factors for acute pneumonia infection are related to disruptions of natural pulmonary host defense mechanisms (104). Cigarette smoke, preexisting disease, alcohol, certain medical procedures bypassing the upper airways, and some commonly prescribed drugs are examples of factors that may compromise pulmonary defense mechanisms and increase host susceptibility to pneumonia (104). Primary viral pneumonia most often occurs in pregnant women and individuals with cardiovascular disease but can also occur in healthy young adults (103). Secondary bacterial pneumonia most commonly occurs in persons over age 65 who have a chronic disease (103). Both types of pneumonia can occur as a complication of influenza. Persons at greatest risk for death due to pneumonia and influenza are infants, older persons, and those with preexisting diseases (102). Although persons with HIV infection are at higher risk of developing and dying from pneumonia, these deaths should be coded to HIV infection as underlying cause of death. It is not known to what degree this potential bias affects the maps shown in this atlas.

Rates are high in East South Central States for older whites, similar to the pattern seen for lung cancer, heart disease, and COPD. In addition, rates are high in the Pacific region for older white females and in the South Atlantic-North region for older white males. Rates for blacks and whites appear high in several Pacific and Mountain-South States, particularly for the younger age group. Previously published maps for these diseases show no elevated rates in these western regions (7), but rates of death due to HIV infection are high in several of these States.

DIABETES

Diabetes mellitus death rates declined from 1950 through the mid-1980's, then increased slightly before leveling off by 1990 (53). For 1988–92, male and female death rates were similar, but the rates for blacks were more than twice those for whites (53). In 1992 diabetes was the seventh leading cause of death among white females, but the fourth leading cause among black, American Indian/Alaskan Native, and Hispanic women (53). The prevalence of diabetes has been estimated as two to three times greater for Mexican Americans and Puerto Ricans than for non-Hispanic whites (105). The mortality figures may underestimate the actual number of deaths due to diabetes by as much as 50 percent, because of failure to note a history of diabetes on the death

certificates of diabetics who die of stroke or heart disease (27).

The etiology of diabetes mellitus has yet to be determined (106). Genetic predisposition seems to play an important role in the development of both noninsulin-dependent and insulin-dependent diabetes mellitus (107). Recent research has suggested that the latter type may be associated with viral infections (106), exposure to certain chemicals and pharmaceutical products (106), and psychological stress (108). The primary risk factor for noninsulindependent diabetes seems to be obesity (107). Differences in socioeconomic factors and higher prevalence of obesity may explain part of the higher risk of diabetes among Mexican Americans compared to non-Hispanic whites (105).

Death rates among whites are highest in the Middle Atlantic and East North Central States, and in southern Texas and New Mexico, similar to patterns seen on maps for 1968–71 (7). The southwestern excess may reflect the high proportion of Mexican Americans living in this region. Rates among blacks are highest along the Gulf and south Atlantic coasts.

Suicide

Since 1980 overall suicide rates have remained relatively stable, with slight increases due primarily to increasing rates among older persons (53, 109). Men are about five times more likely than women to commit suicide, and rates among whites are twice those among blacks. Native American males have the highest suicide rates of all races up to age 44, but for older ages, white males have consistently had the highest rates (53). Firearms are the most common method of suicide used by men and women, although a drug overdose is the method of choice for nearly as many women (109).

Persons with a family history of suicide, alcohol abusers, and individuals diagnosed with a psychiatric illness (for example, depression) or a personality disorder are at a higher risk of suicide (110, 111). Other possible risk factors include the level of certain neurotransmitter metabolites, previous suicide attempts, stressful life events such as divorce or widowhood (109, 110), and firearm availability. The observation of suicide clusters among young persons has suggested that exposure to a suicide may initiate suicidal behavior (110, 111).

Geographic patterns for suicide rates vary by method of suicide (95), but rates for all types of suicide combined are highest in the western States (12, 95) and in nonmetropolitan areas. See cautionary note in "Amended data" section under "Causes of death."

FIREARM SUICIDE

During 1988-92 firearms were associated with 65 percent of all suicides among men and 42 percent among women; although, among women under age 75, the most common method of suicide is by firearm. The trends in firearm suicide rates paralleled the overall suicide rates through the 1960's (111); but between 1968 and 1986, the firearm suicide rate increased 36 percent, while suicide rates for other methods remained stable (95). As with all suicides, rates for whites are several times those for blacks, but an even greater gender difference is seen for firearm suicide, with the highest rates among older white males. The firearm suicide rate among adolescents has doubled in the last 30 years (112), now accounting for two-thirds of all suicides among persons ages 15-34 years (113).

Risk factors are the same as for all suicides as noted in the previous section.

The highest rates of suicide by firearms are found in the Mountain regions and scattered southern States, where gun ownership has been more common (95). Among whites high rates predominate in the Mountain and West South Central regions for younger males, while the East South Central, South Atlantic, and Pacific regions have higher rates among older males and among females. In all regions of the United States, firearm suicide rates are highest in nonmetropolitan areas.

LIVER DISEASE

After peaking in the early 1970's, chronic liver disease and cirrhosis death rates have steadily declined by about 3 percent annually through 1992 (53). During 1988–92 death rates were about twice as high for males compared to rates for females and for blacks compared to whites. Excess deaths from cirrhosis have been noted among Mexican-born Americans (114). Of all deaths in this category, 79 percent were due to cirrhosis, 10 percent to an unspecified liver disease, 6 percent to hepatitis, and 5 percent to other liver diseases.

Heavy alcohol use is considered a major risk factor for chronic liver disease (115) and is a well-established risk factor for cirrhosis (116). Younger men are more likely to consume alcohol than older men (53). However, among drinkers, younger black men and older Hispanic men are more likely to have

heavier consumption than others (53). Infection with hepatitis B or C virus is also believed to increase risk of chronic liver disease, as are certain drugs, industrial chemicals, and less common infectious agents (115). Liver cirrhosis may develop as a result of an interaction between alcohol consumption and other risk factors (117). Established risk factors for chronic hepatitis include viral infection, autoimmune disease, drug use, and certain genetically transmitted metabolic disorders (118).

Among whites rates are highest in the Mountain-South and Pacific regions; rates for the younger ages are particularly high for the Pacific region. Cirrhosis mortality among whites was high in California during 1965–71, but most rates in the Mountain-South region were not significantly high at that time (7). High rates among blacks are seen in scattered areas throughout the south and west.

Human immunodeficiency virus infection

Death rates due to HIV infection nearly doubled over the period covered by this atlas, from 6.7 percent in 1988 to 12.6 percent in 1992 (53). The highest rates occur in the 25 to 44-year age group, accounting for 73 percent of all HIV infection deaths (119). Substantially higher HIV infection and death rates occur among males and among blacks (53, 120). Black male death rates for 1988–92 were triple those of white males; more black men died in 1992 from HIV infection than from unintentional injuries or stroke (53). Since 1988 death rates among females have risen slightly faster than death rates among males, but rates among females are still much lower than rates among males.

HIV infection is primarily spread through a number of high risk sexual practices and the sharing of contaminated drug paraphernalia (121). Though anyone who practices these high risk behaviors is at risk, persons recognized to be at greatest risk for HIV infection include homosexual and bisexual men, injecting drug users, and females who have sexual contact with bisexual men or injecting drug users (120–122). Other populations at risk for HIV infection include infants born to HIV-infected mothers and hemophiliacs and others exposed to contaminated blood or blood products through transfusion or occupational activities (121).

The geographic distribution of new acquired immunodeficiency syndrome cases reported for 1993 indicates that the majority of the cases are concentrated in major metropolitan areas, particularly

in Florida, California, and the urban corridor from Washington, D.C., to Boston, Massachusetts (123). A similar, but less concentrated, pattern is seen for death rates, with few notable differences between maps for males and females or for younger and older ages despite the large differences in rates for these groups.

Homicide

Since the mid-1950's, homicide rates have generally increased (95), rising about 5 percent annually from 1987 to 1991, followed by a 4-percent decline to 1992 (53). Historically, these trends have reflected changes in firearm homicide rates, as rates for other types of homicides have remained relatively stable over time (95). One-third of all homicides occur among persons ages 15-24 years (53). Homicide is the leading cause of death among black males in this age group (53). For black males of all ages, homicide ranks third of the causes included in this atlas, ahead of unintentional injuries and stroke. Black male rates are higher than those for black females and white males and females for every age group. Mortality statistics for 1993-94 suggest that rates may have peaked in 1992 (124).

Factors that influence the risk of being a victim of homicide include age, sex, ethnicity, and socioeconomic status (125). Other factors hypothesized to be associated with homicide are firearm availability, alcohol and drug use, drug trafficking, racial discrimination, and cultural acceptance of violent behavior (126).

For all race/sex groups, the highest homicide rates are seen across the southern States; the extension of high rates into the Pacific region for

whites was less apparent in maps that combined data over racial groups (12, 95). For all ages and races, 1992 homicide rates were nearly twice as high in metropolitan regions as in nonmetropolitan regions and are particularly high for blacks in urban areas. See cautionary note in "Amended data" section under "Causes of death."

FIREARM HOMICIDE

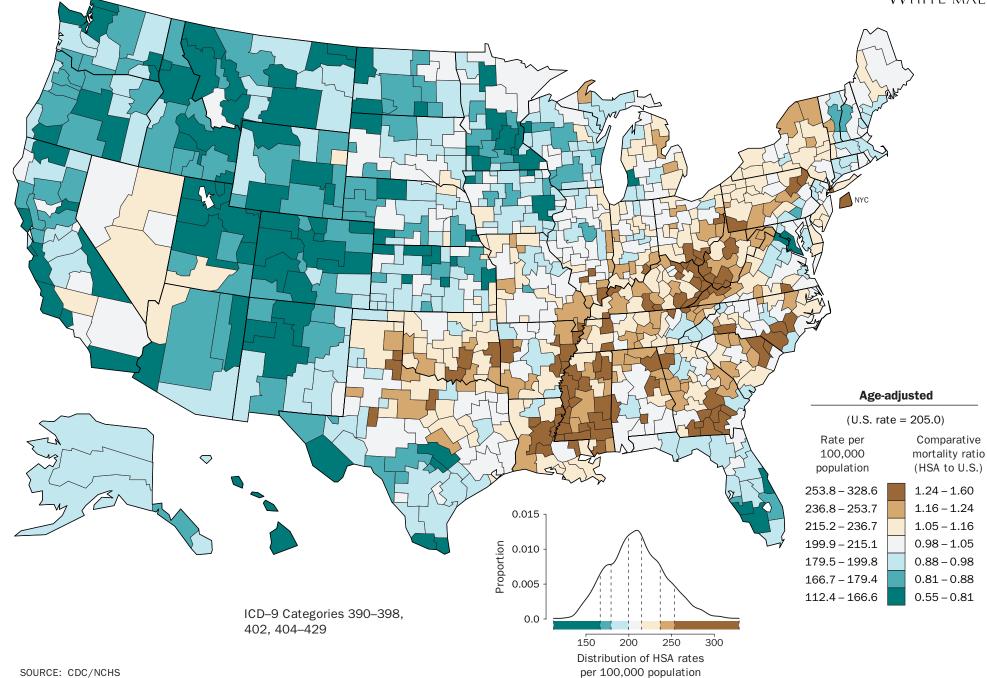
While the death rates from other types of homicide remained relatively stable, firearm homicide rates varied greatly from 1930 to 1986 and have consistently influenced overall homicide trends (95, 124). During 1988–92, a firearm was used in the commission of 65 to 75 percent of all homicides among males and 50 percent of all homicides among females. Firearm homicide rates have been rising most significantly among adolescents and young adults (124). From 1985 to 1990, the firearm homicide rate among black teenage males nearly tripled, while doubling among white males and black females (113). As with overall homicide rates, the rates for black males are much higher than those of any other race or sex for each age group.

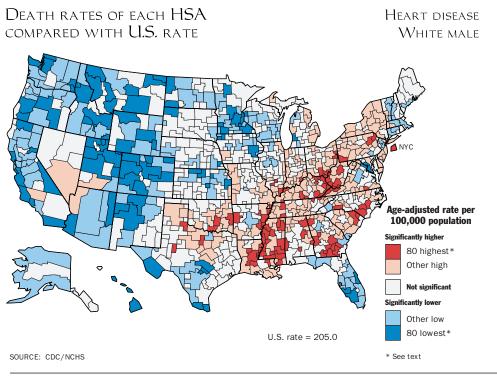
The risk factors and geographic patterns for firearm homicide are similar to those of all homicides.

All causes

Patterns on the all causes maps are influenced by patterns of rates for the leading causes of death. The influence of heart disease and lung cancer can be seen as high rates in the East South Central region, and for women, in the Pacific region.

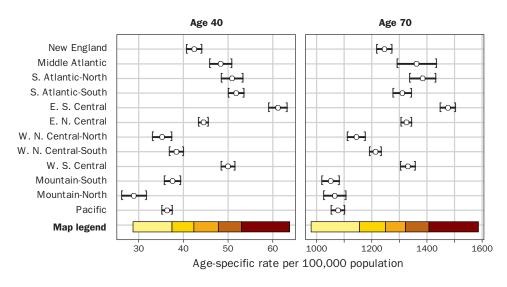
MAPS



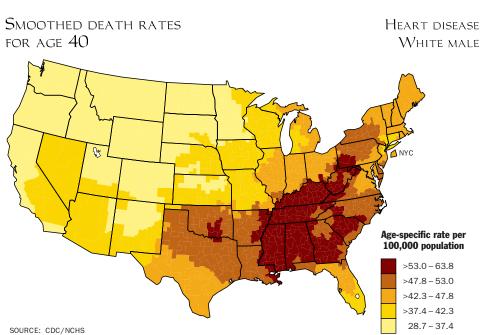


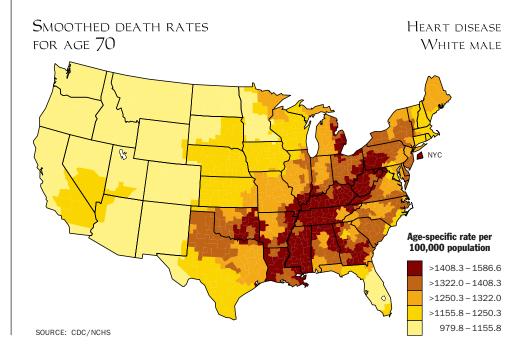
PREDICTED REGIONAL RATES FOR SMOOTHED RATE MAPS

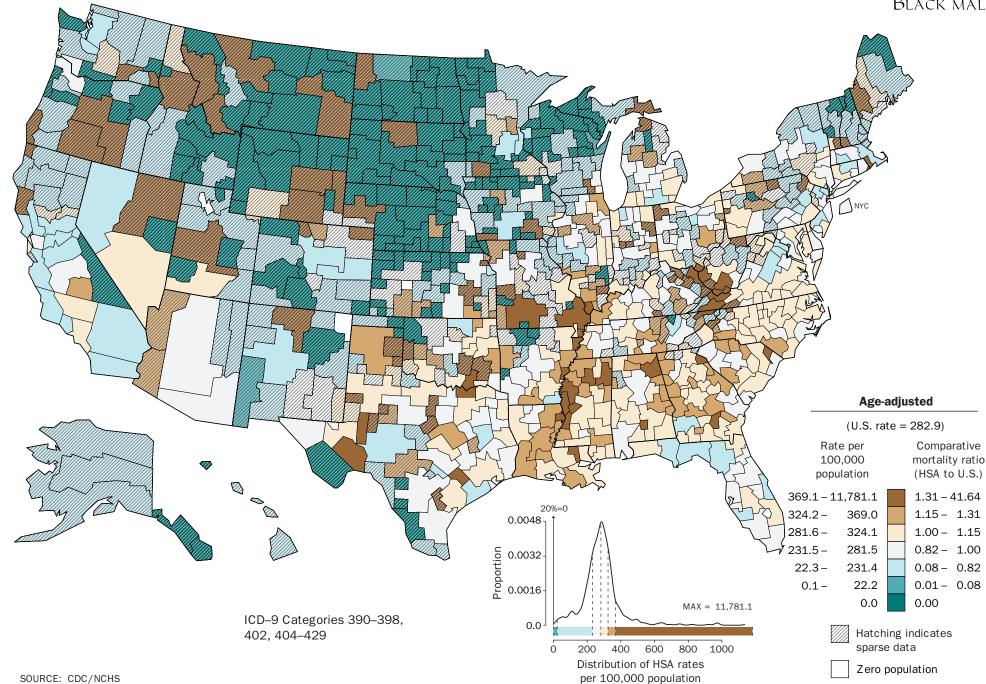
HEART DISEASE WHITE MALE

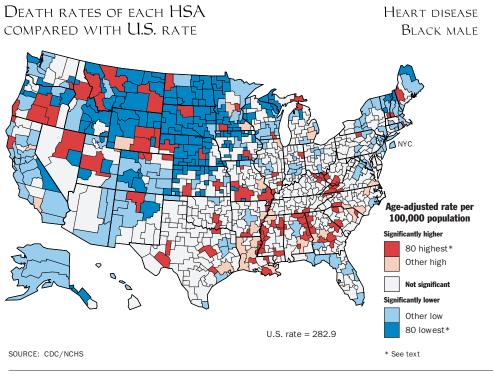


NOTE: Brackets indicate 95% confidence limits. SOURCE: CDC/NCHS



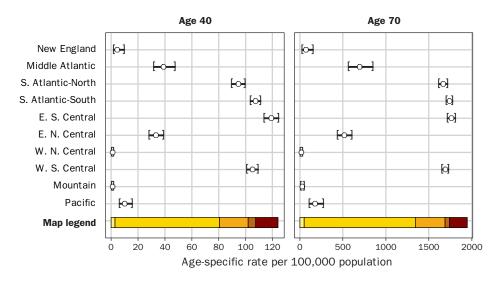


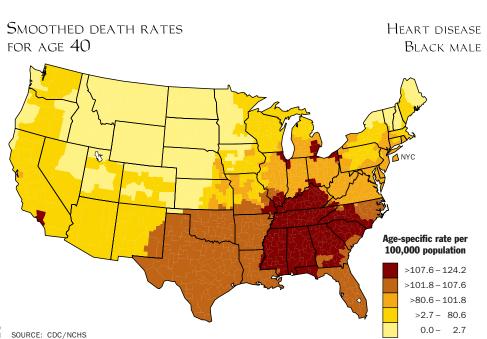


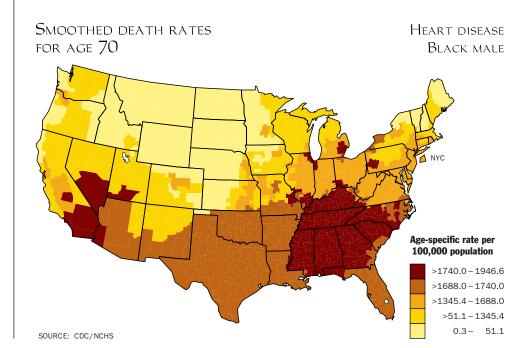


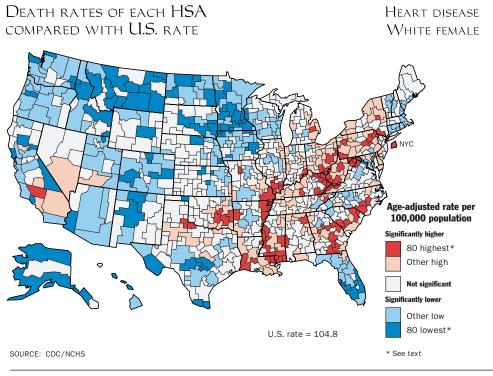
PREDICTED REGIONAL RATES FOR SMOOTHED RATE MAPS

HEART DISEASE BLACK MALE



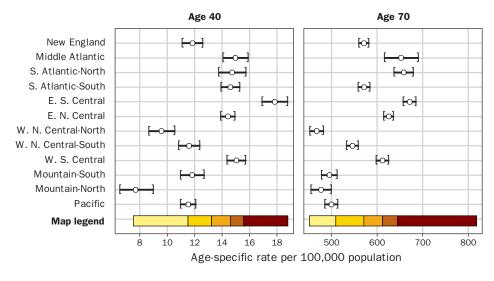


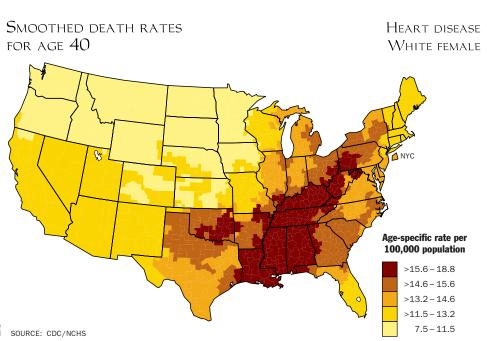


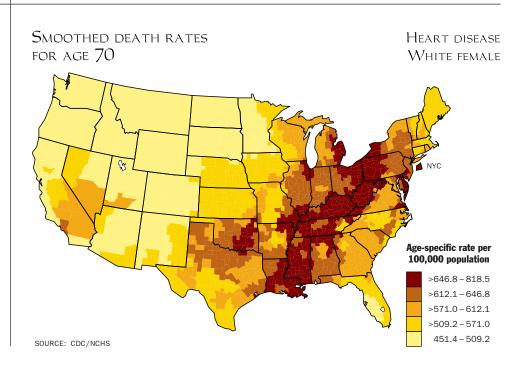




Heart disease White female

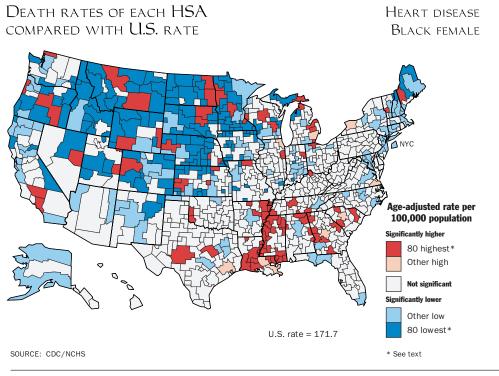






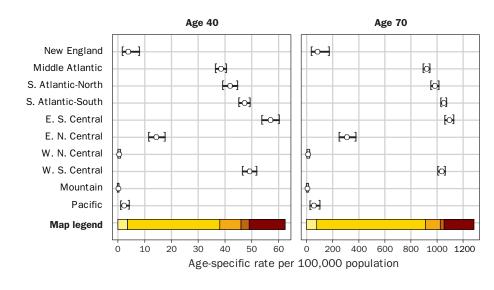
Zero population

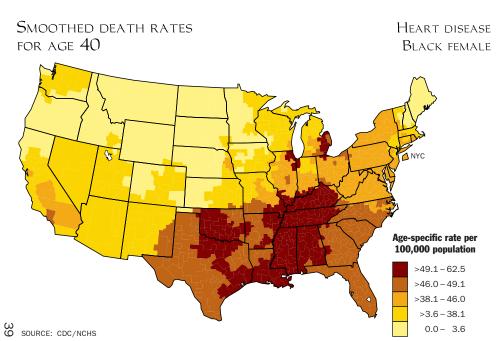
Distribution of HSA rates

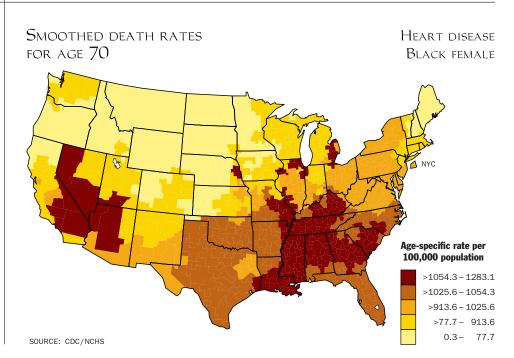


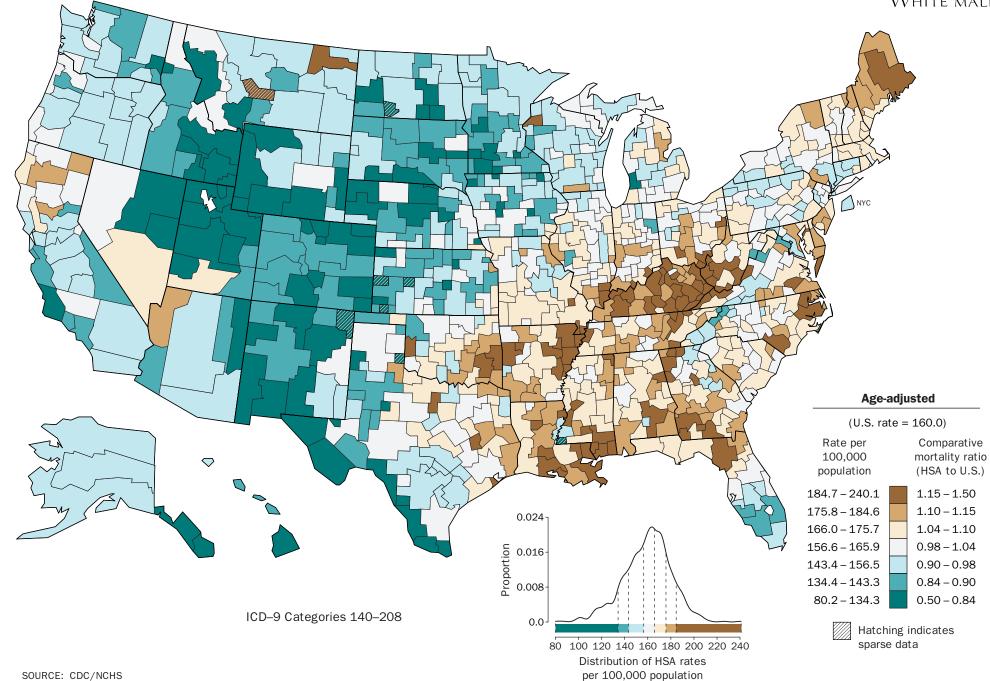
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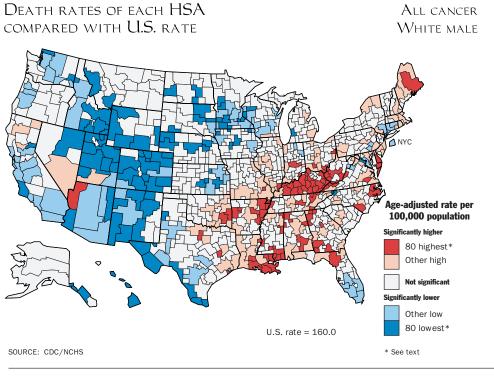
HEART DISEASE BLACK FEMALE





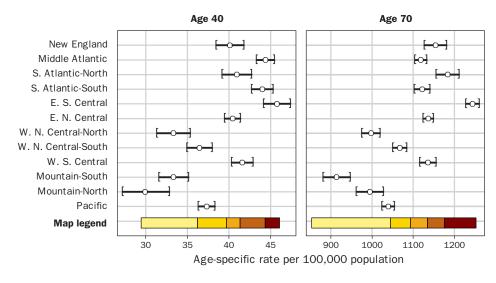


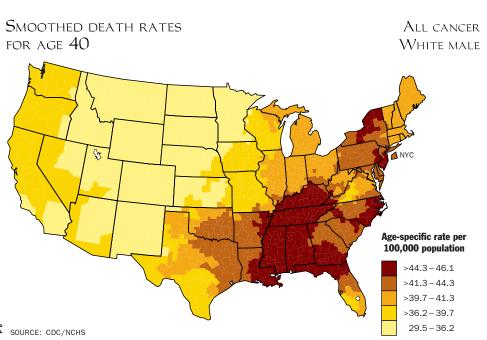


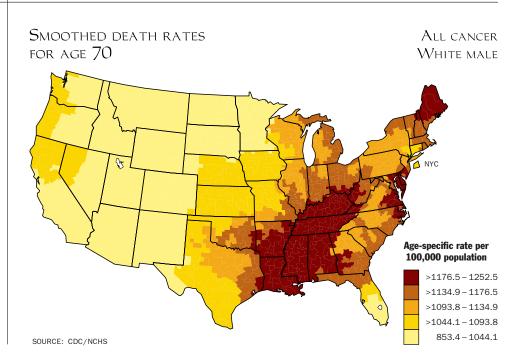


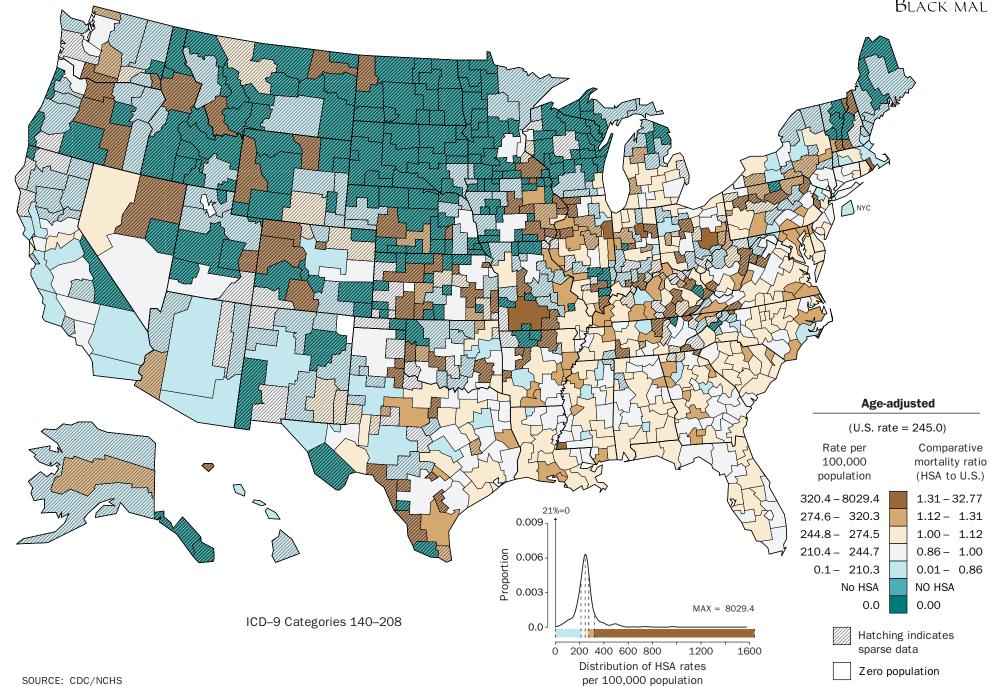


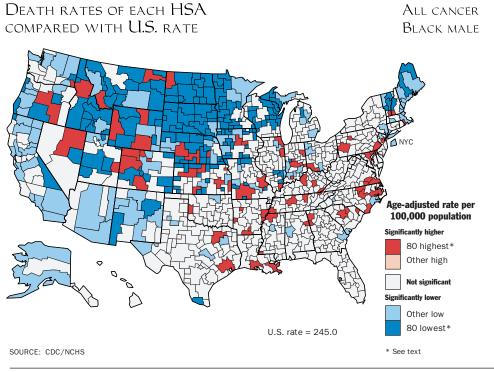






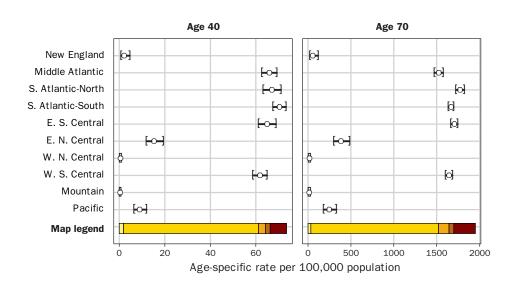


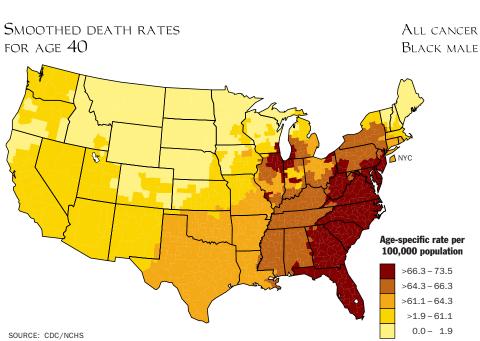


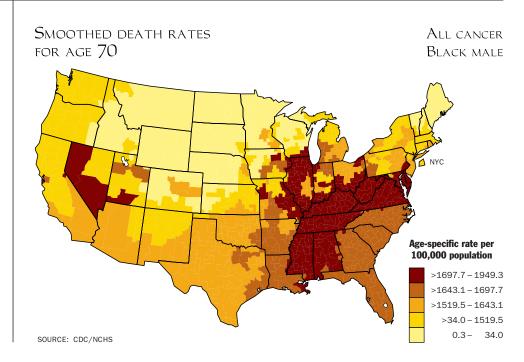


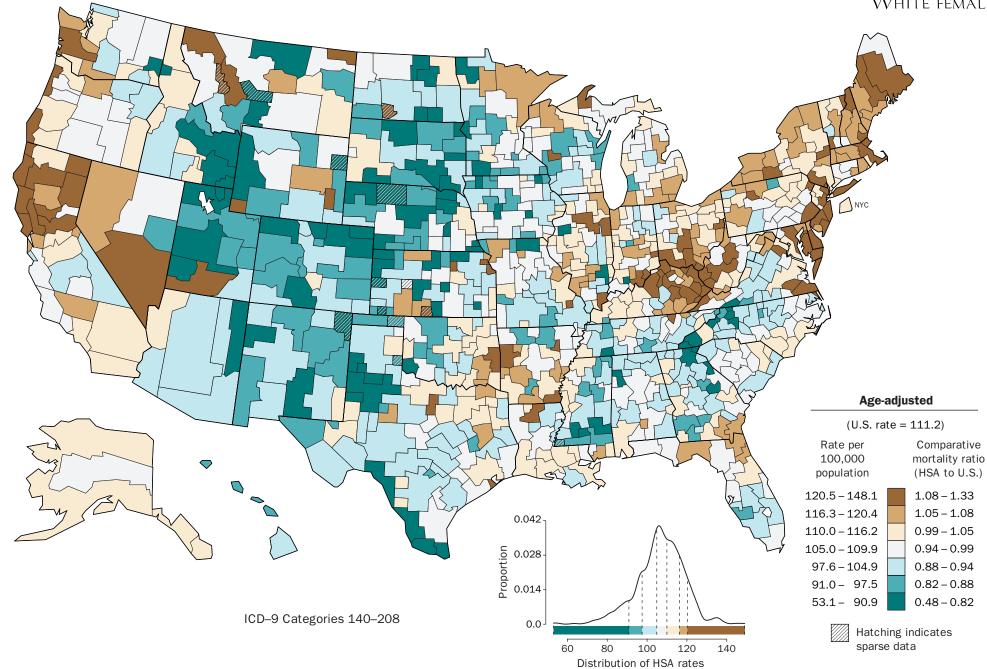


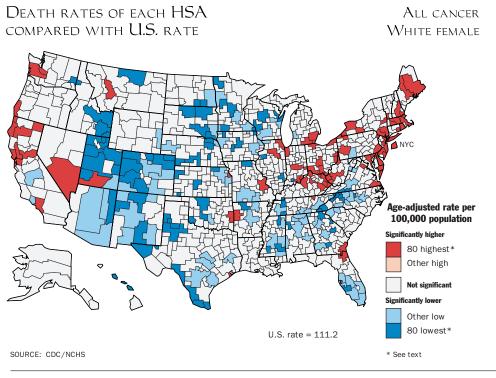
ALL CANCER
BLACK MALE





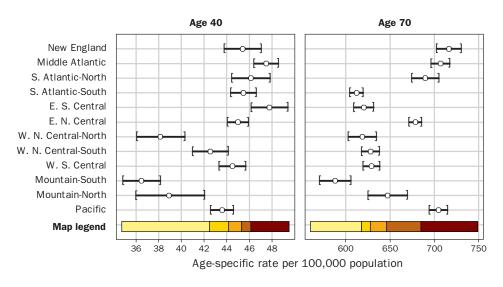


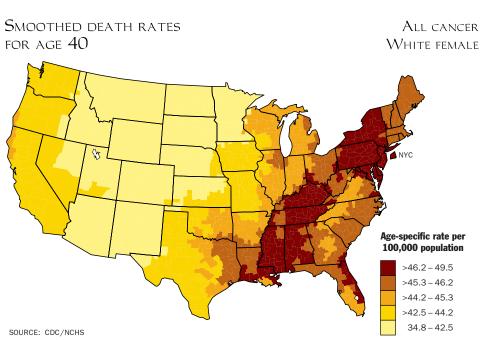


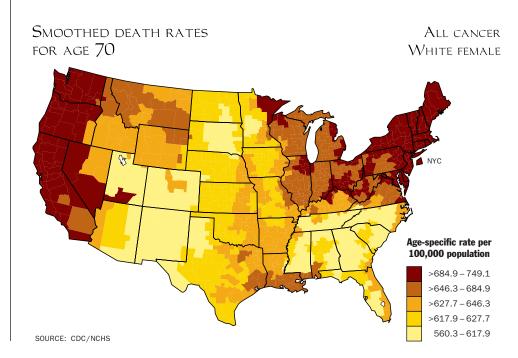


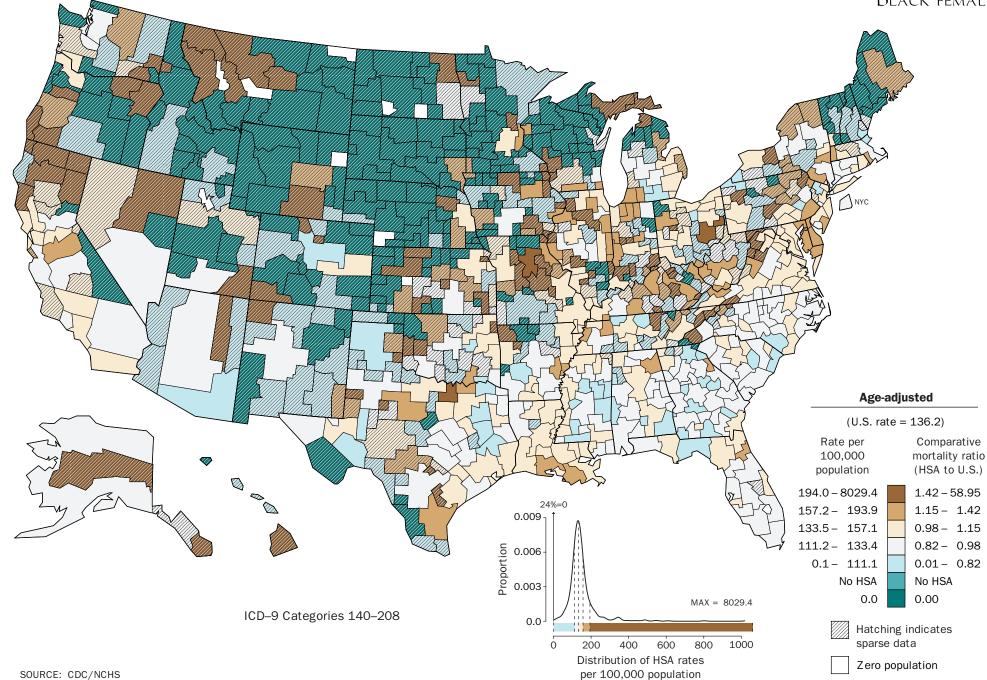
PREDICTED REGIONAL RATES FOR SMOOTHED RATE MAPS

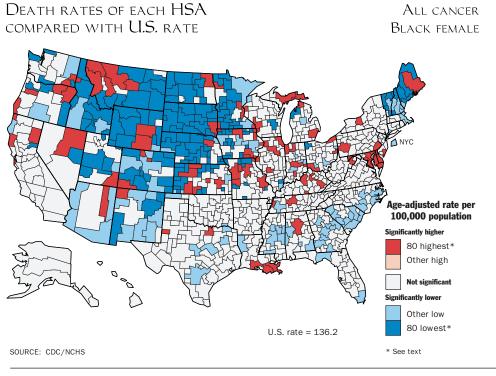
All cancer White female





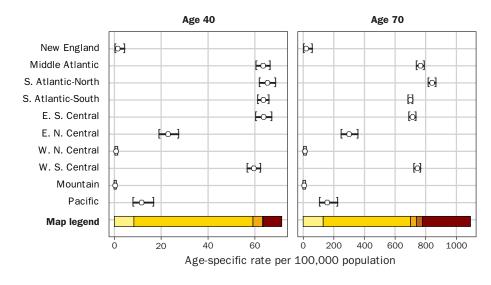


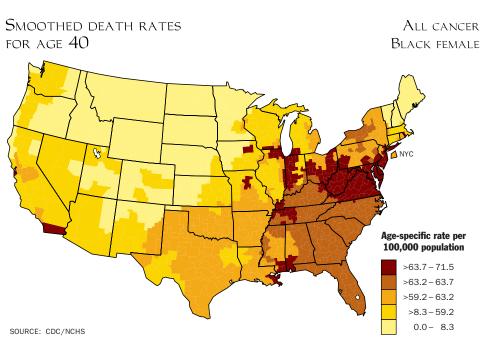


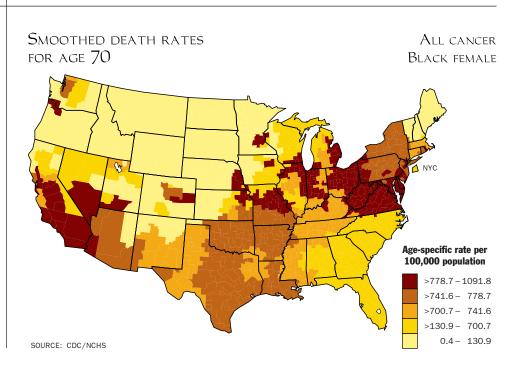


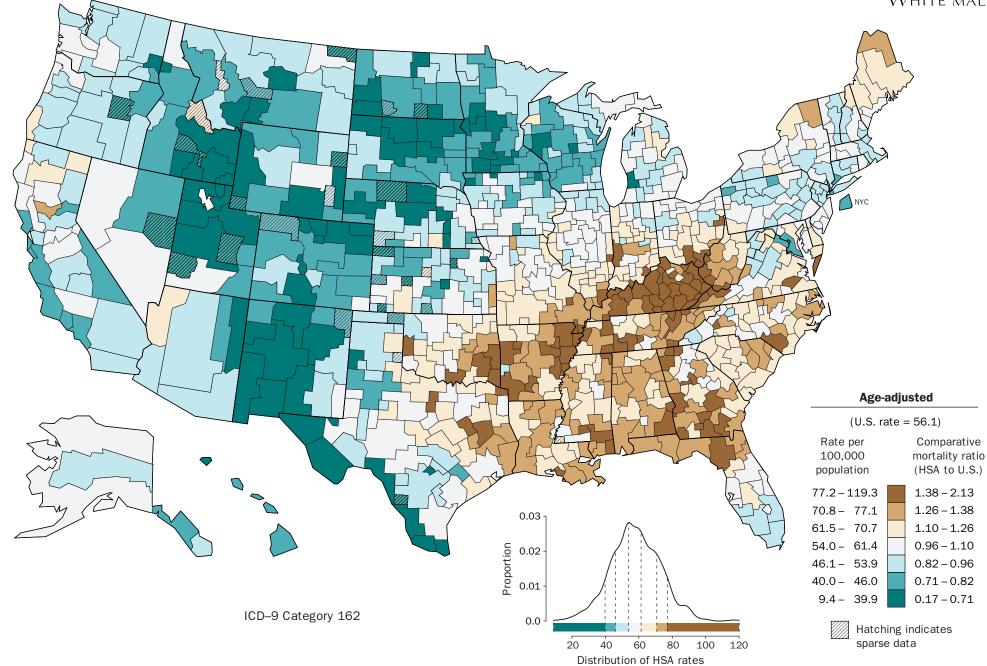
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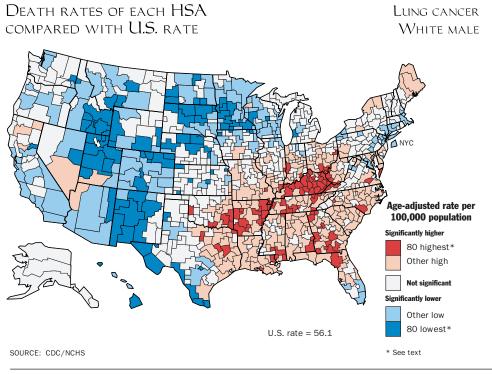
ALL CANCER
BLACK FEMALE





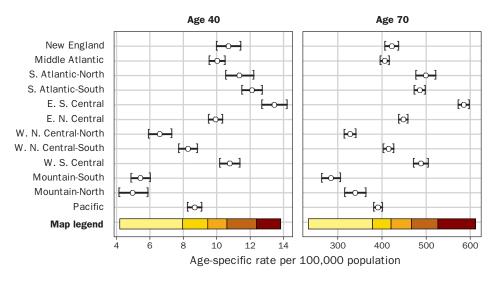


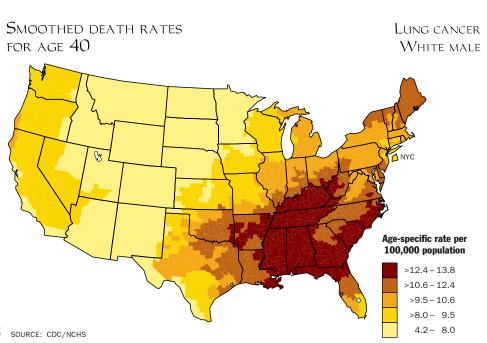


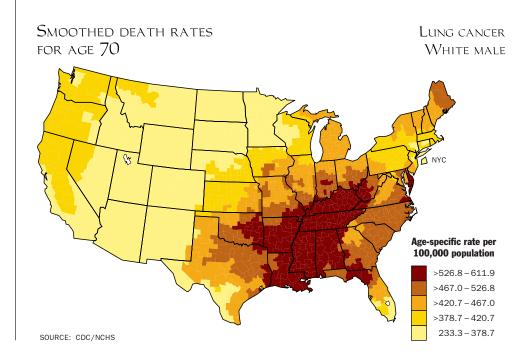


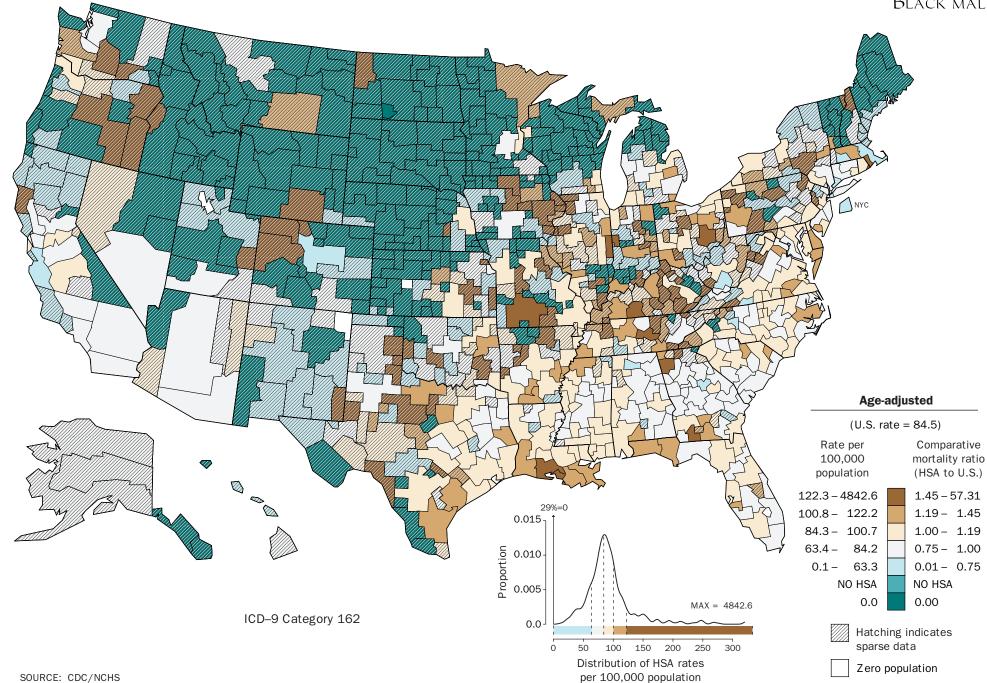
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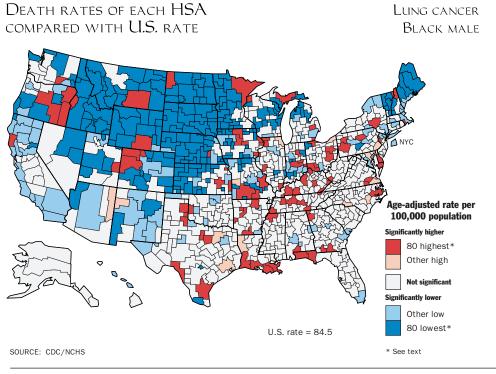
Lung cancer White male





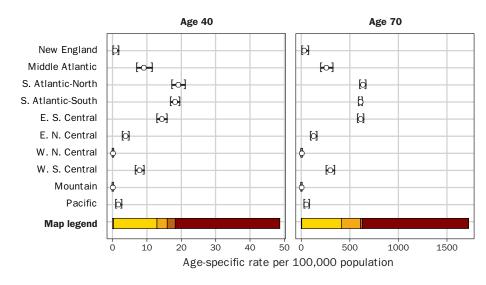


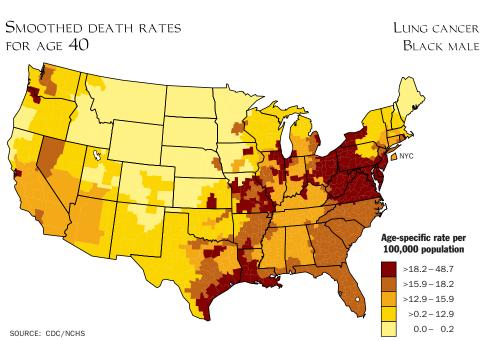


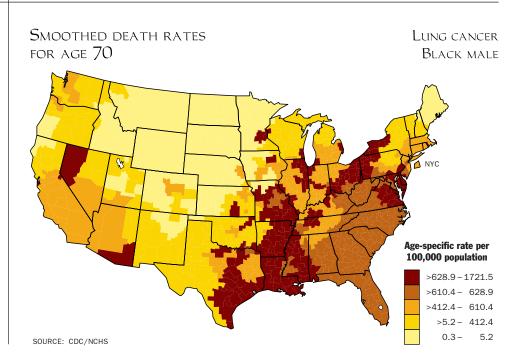


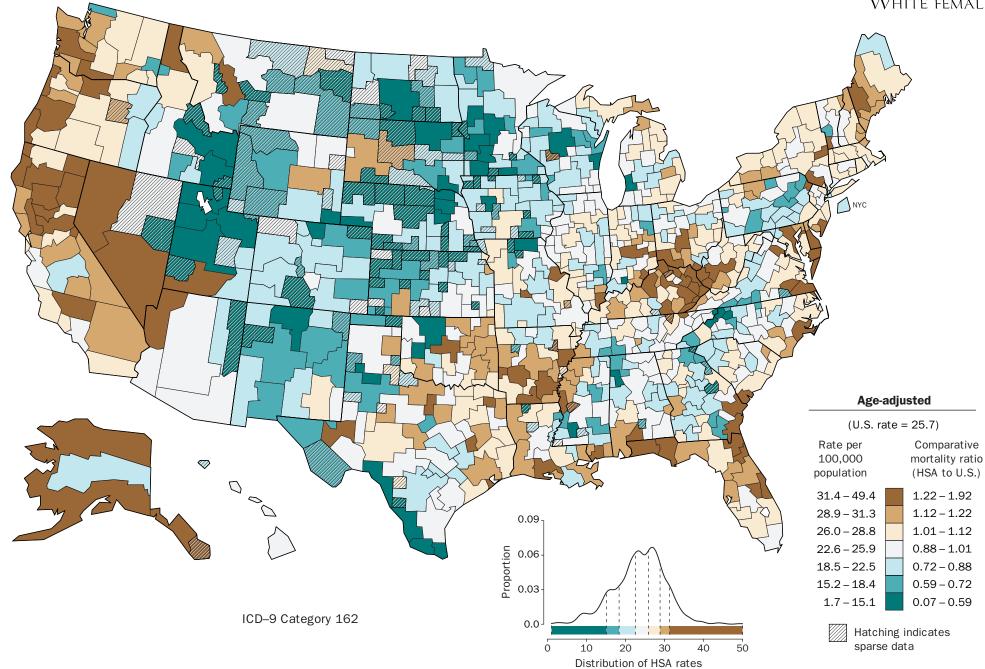


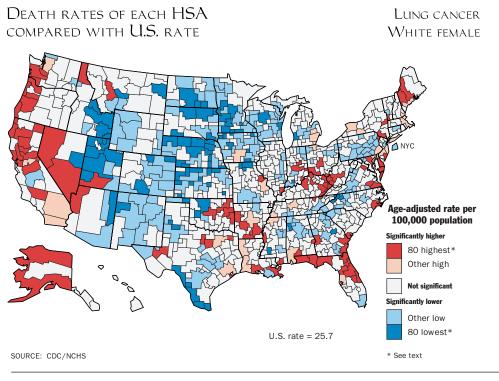
Lung cancer Black male





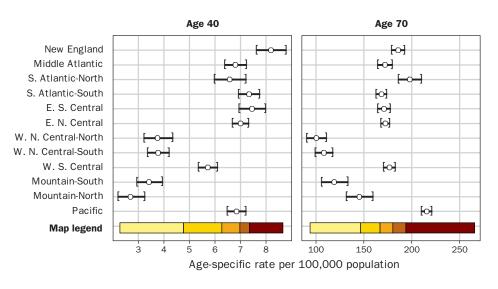


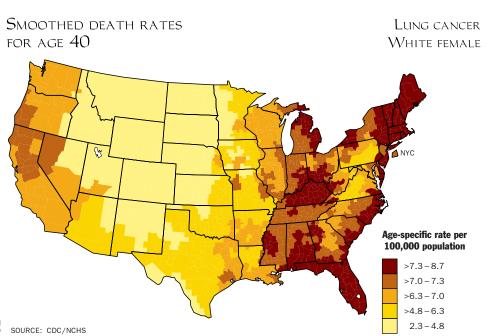


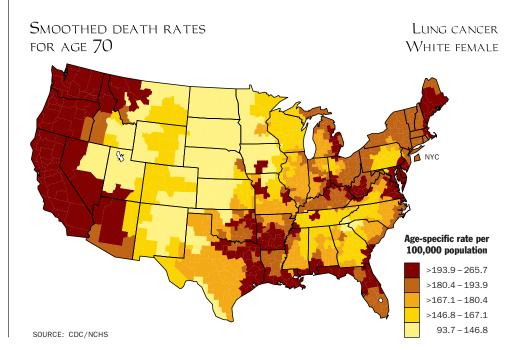


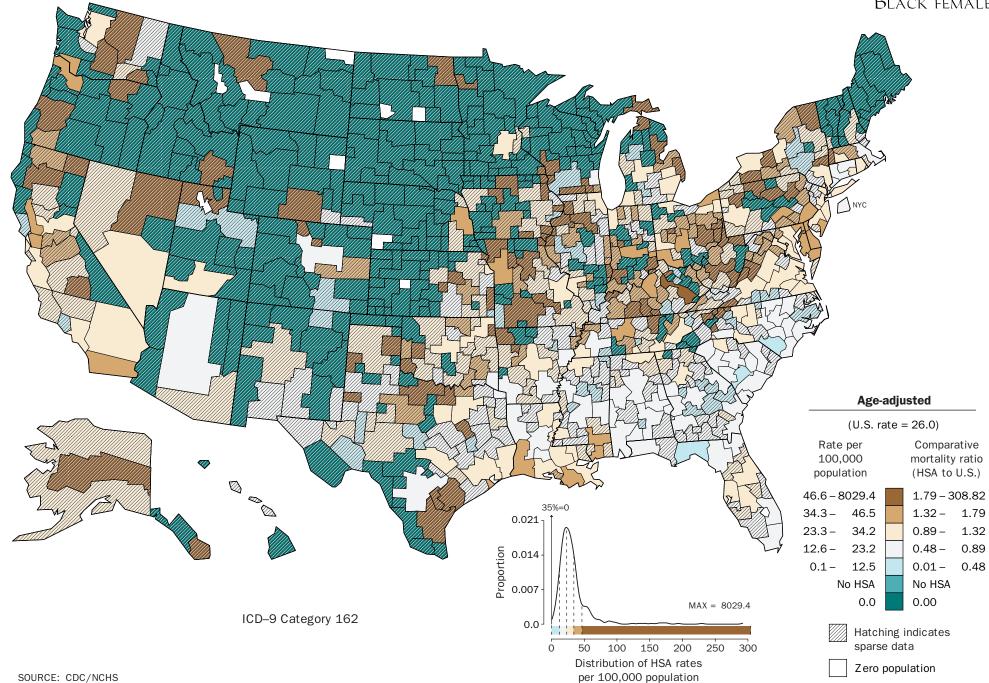
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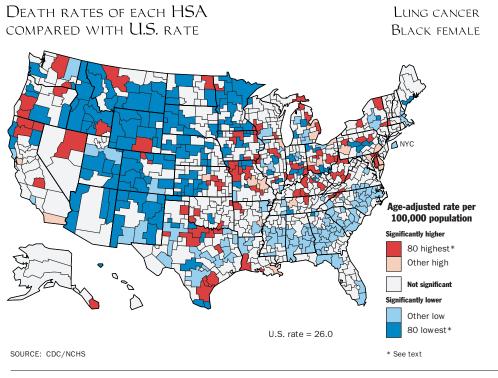
LUNG CANCER White female





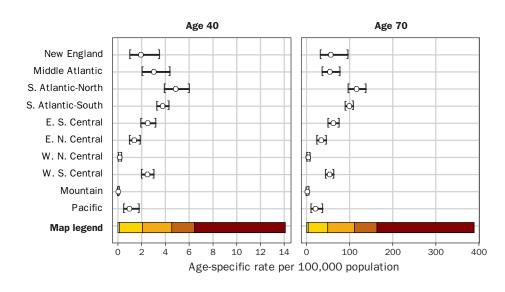


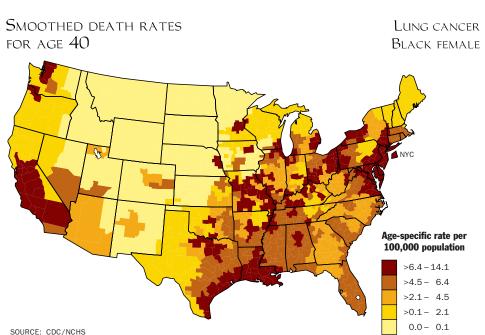


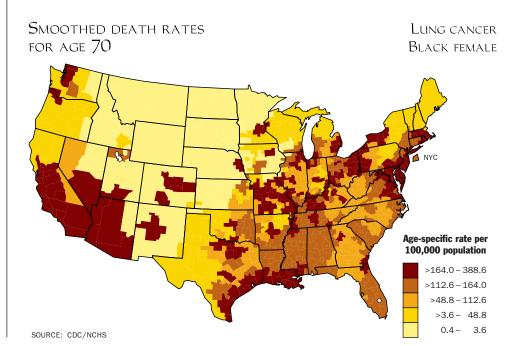


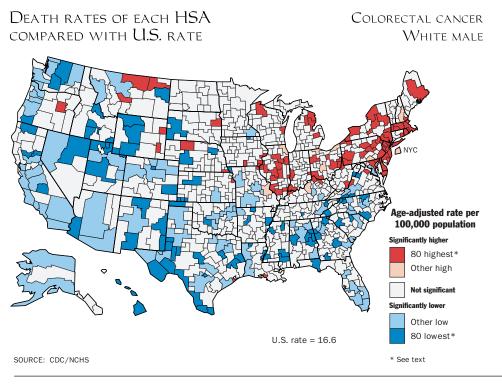
PREDICTED REGIONAL RATES FOR SMOOTHED RATE MAPS

LUNG CANCER BLACK FEMALE



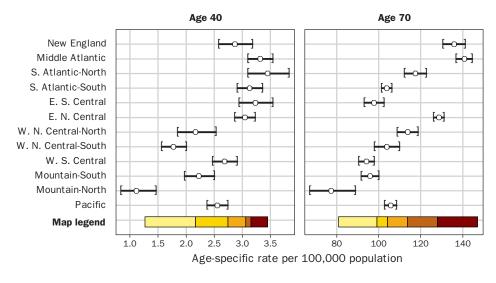


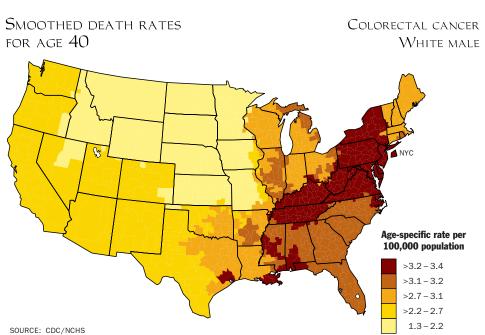


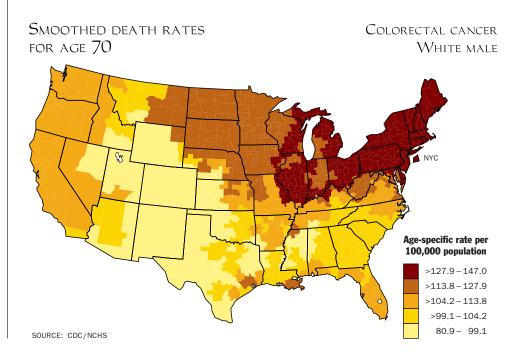


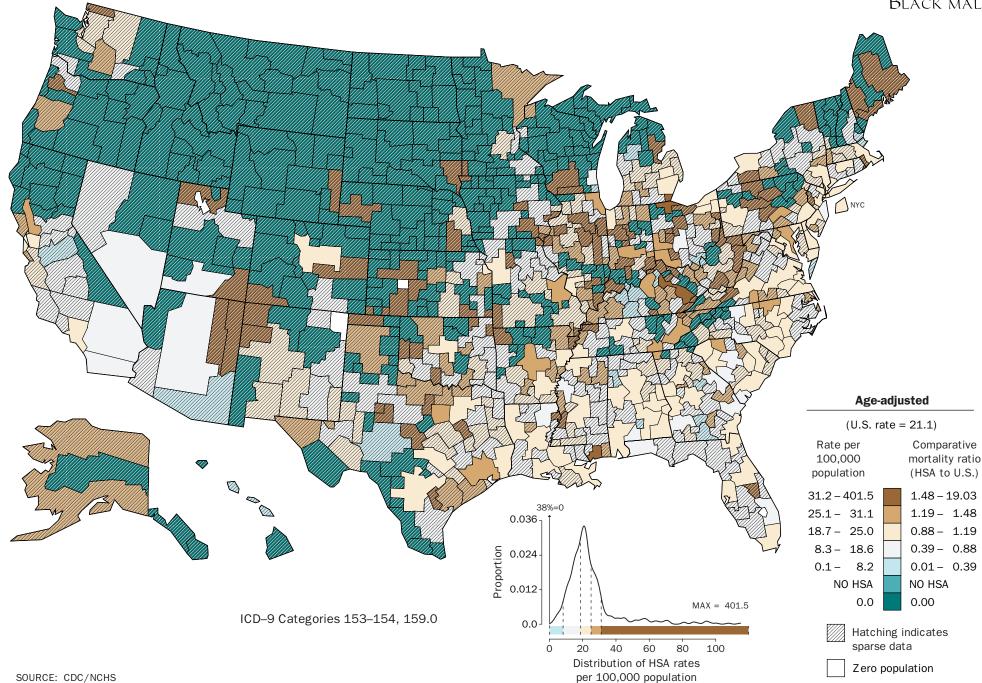
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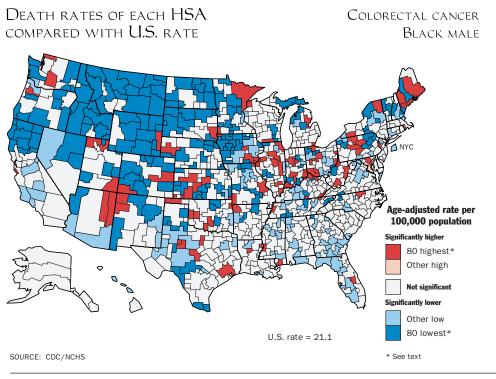
Colorectal cancer
White male





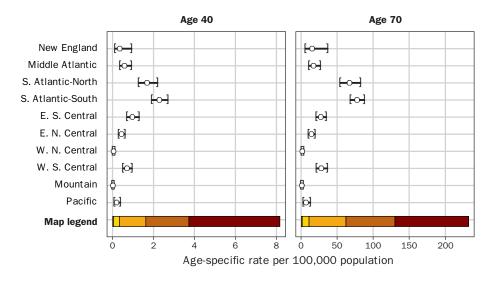


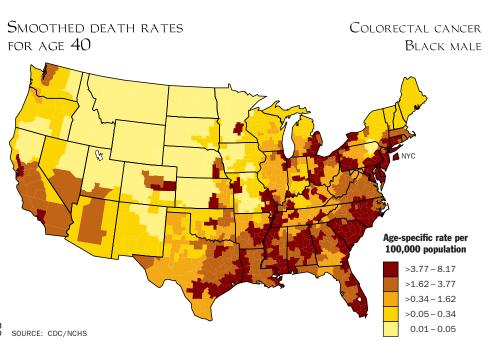


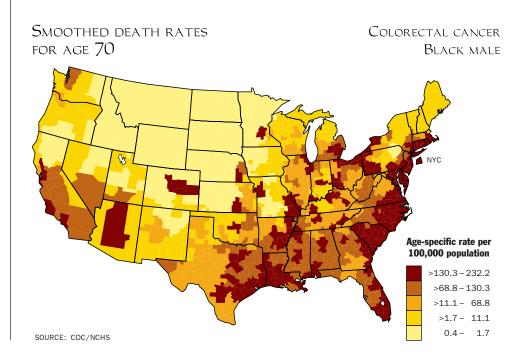


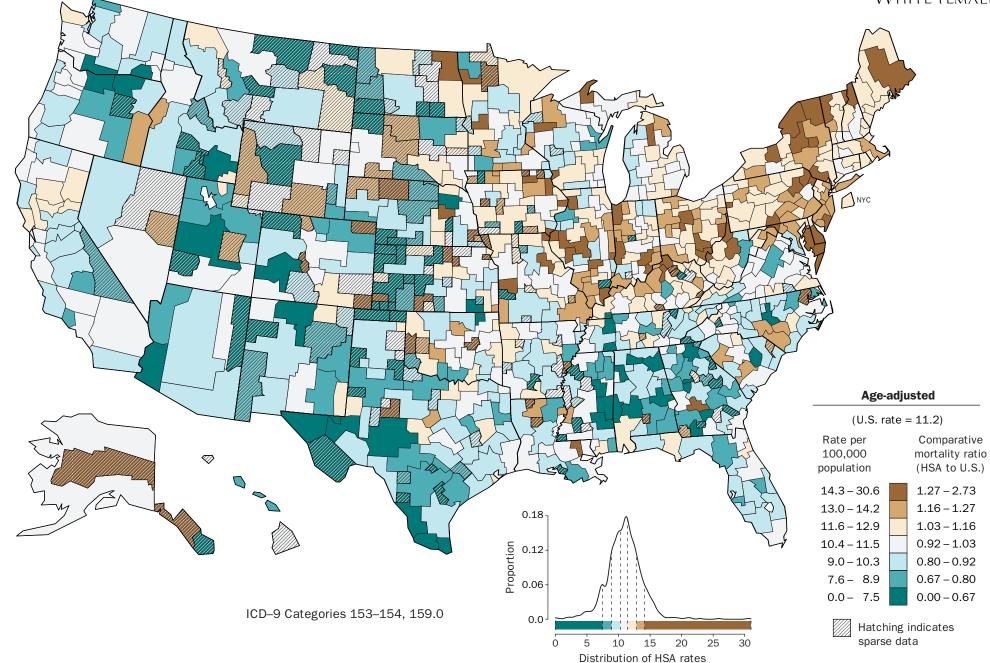
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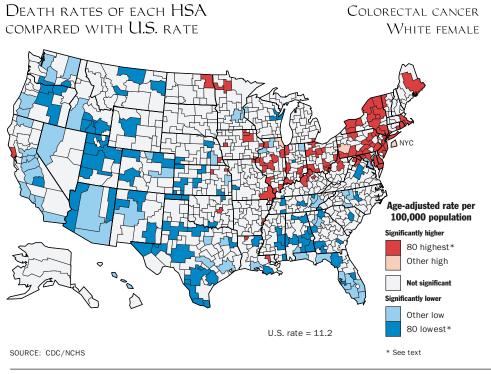
Colorectal cancer
Black male





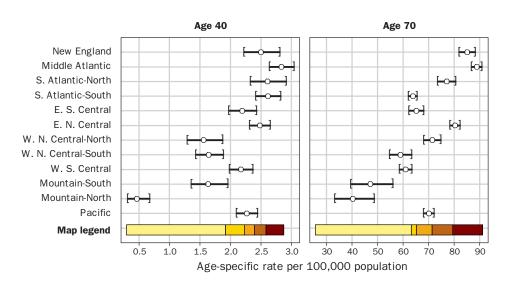


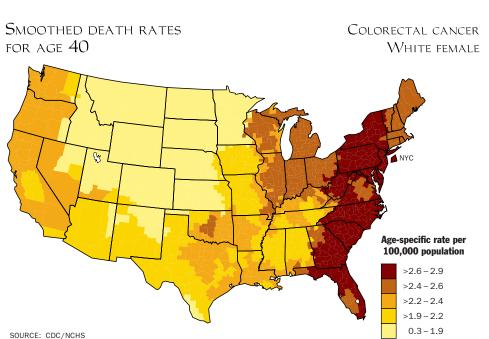


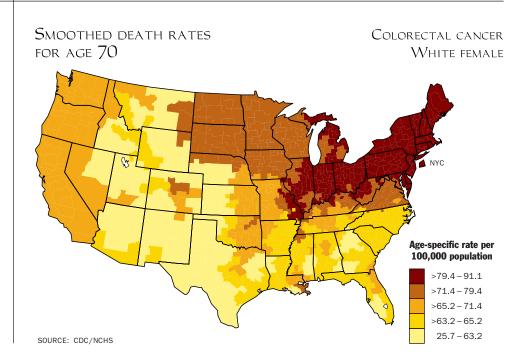


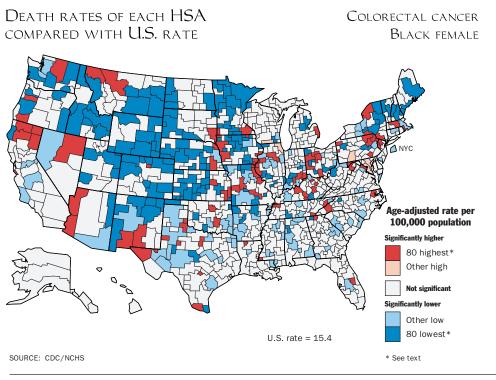
Predicted regional rates FOR SMOOTHED RATE MAPS

COLORECTAL CANCER White female



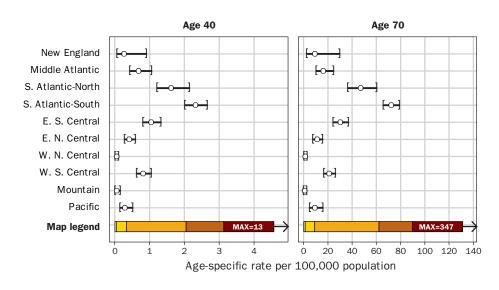


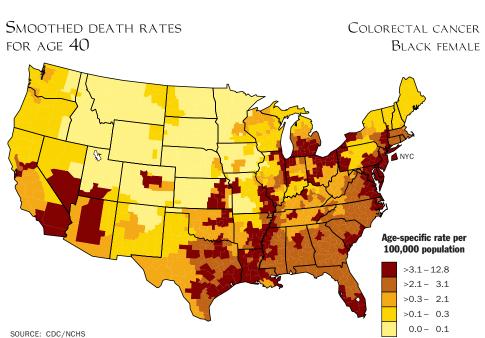


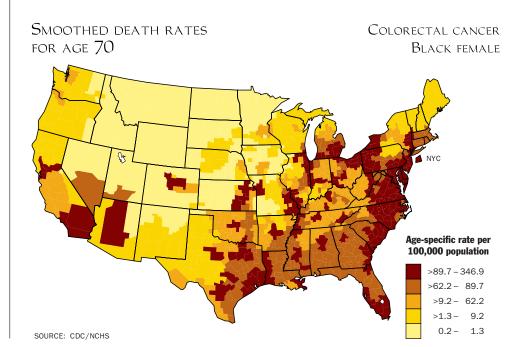


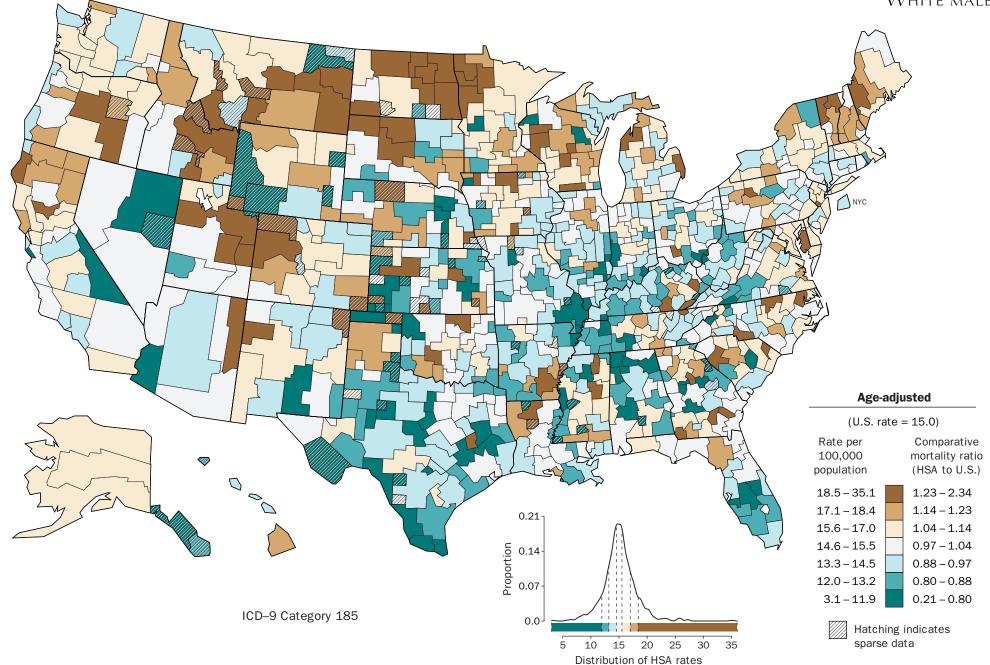
PREDICTED REGIONAL RATES FOR SMOOTHED RATE MAPS

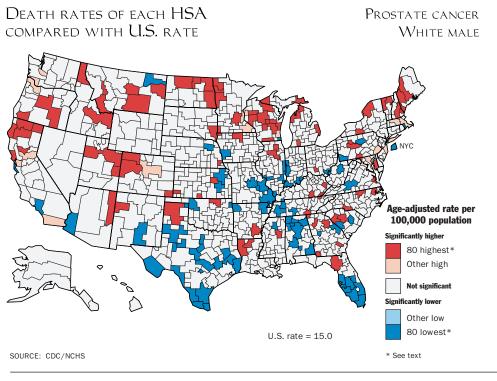
COLORECTAL CANCER
BLACK FEMALE





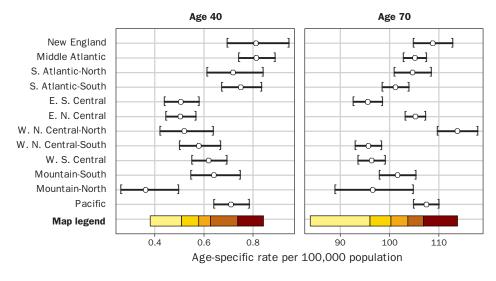


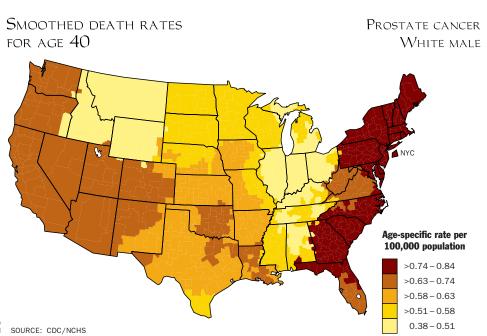


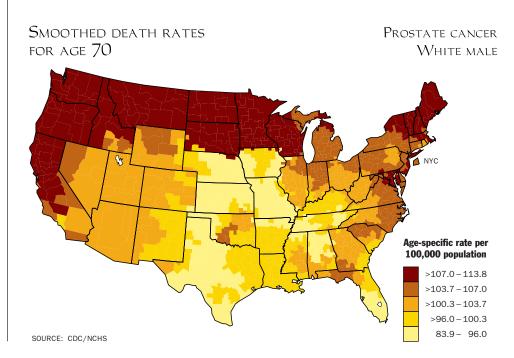


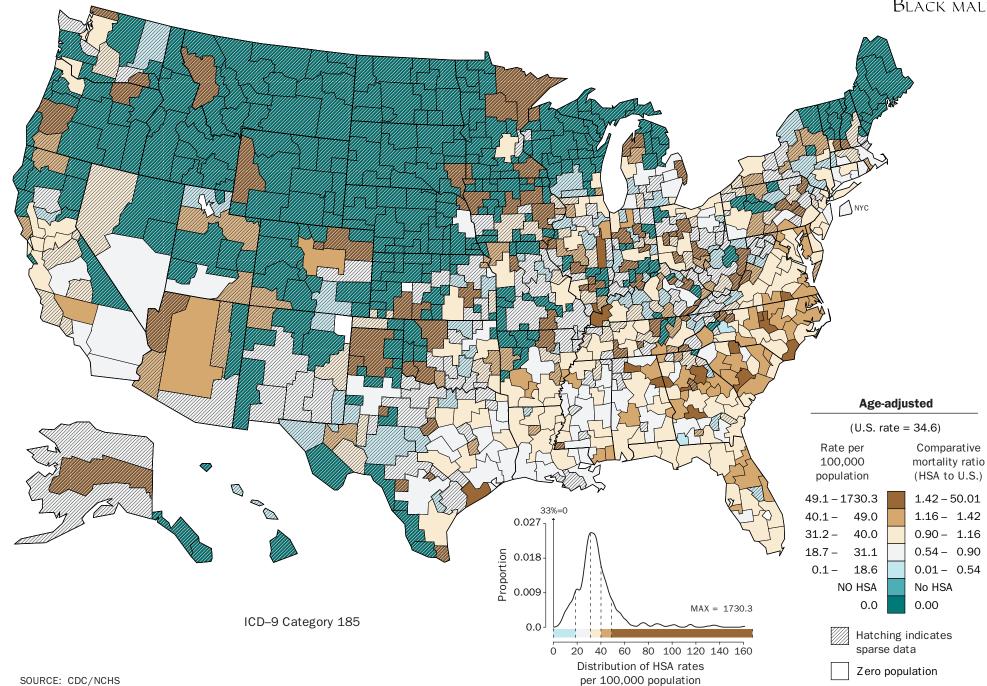


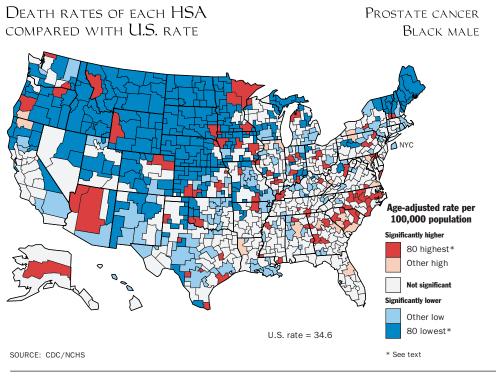
Prostate cancer White male





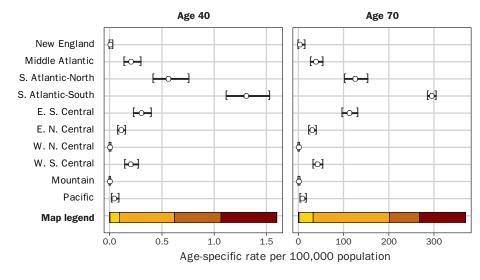


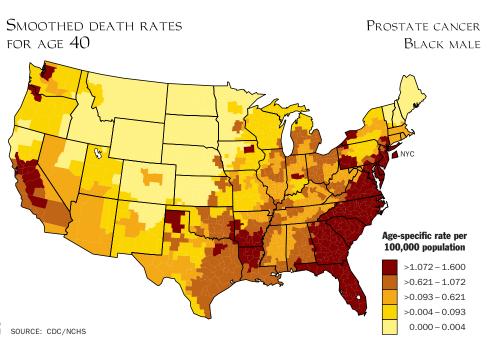


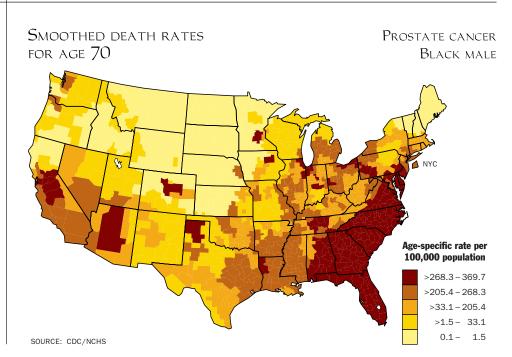


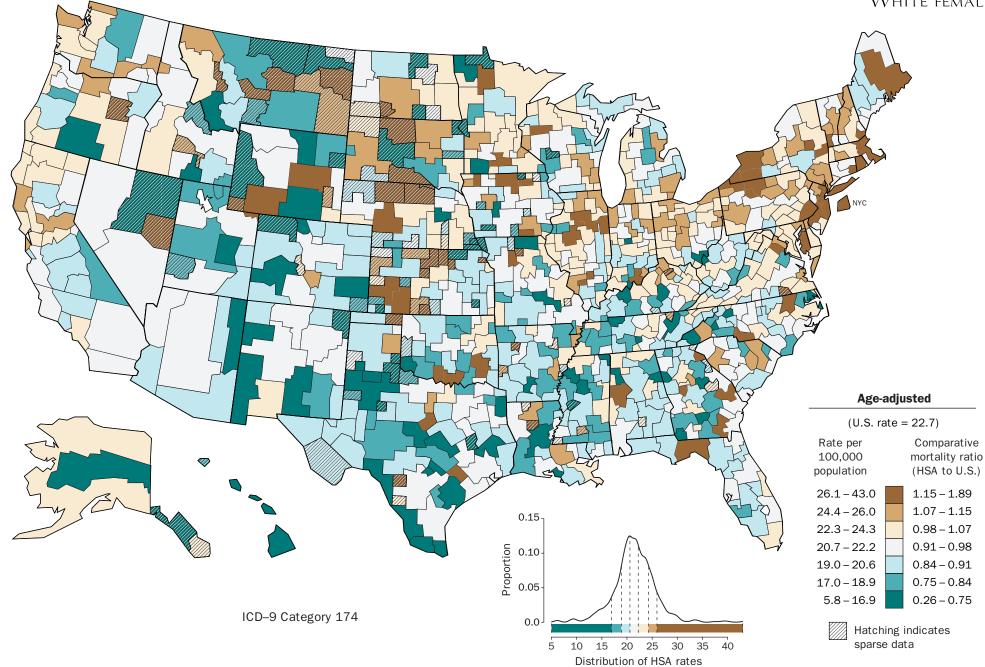


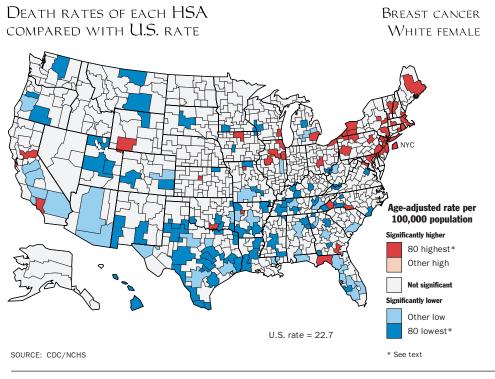
PROSTATE CANCER
BLACK MALE





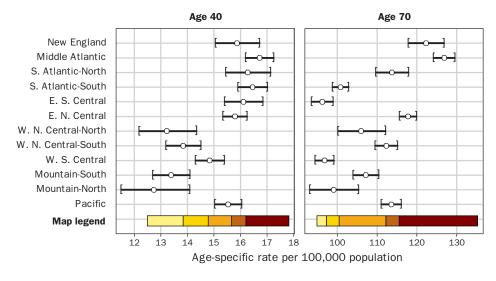


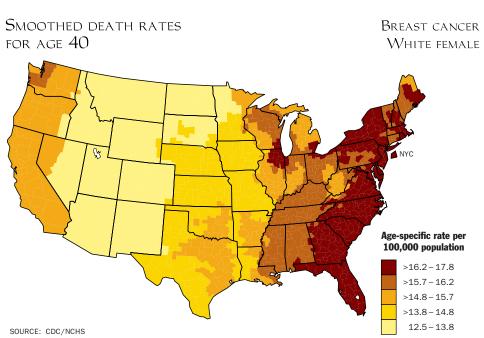


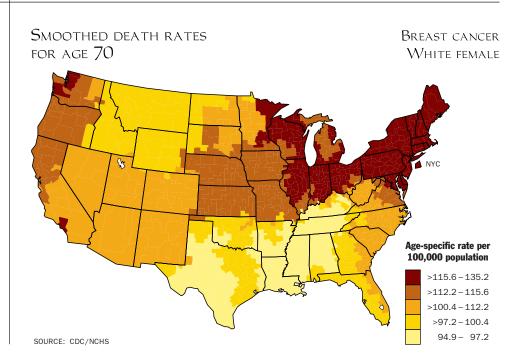


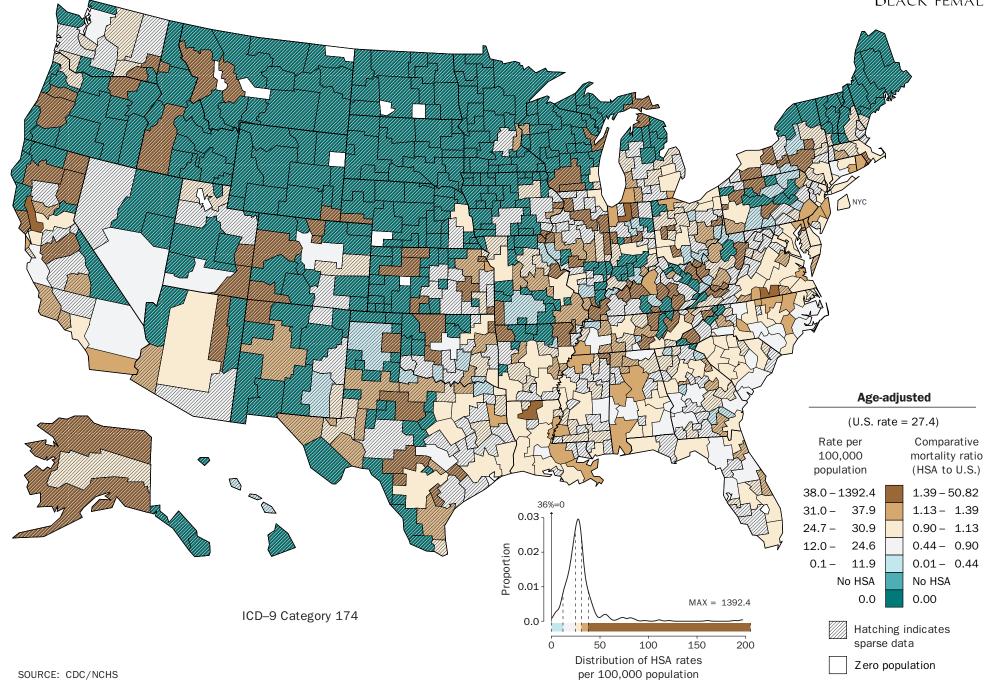


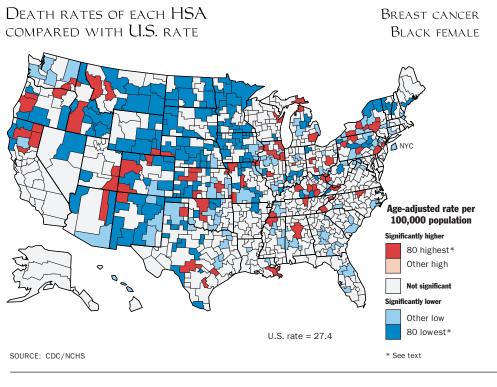
Breast Cancer White female





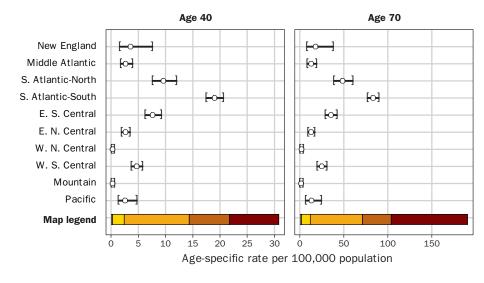


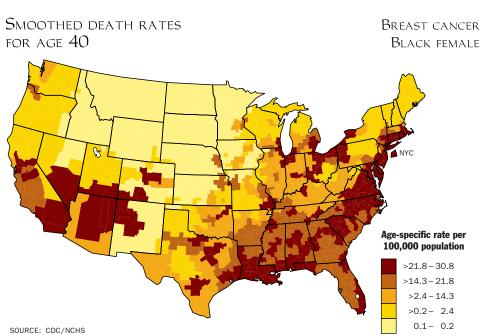


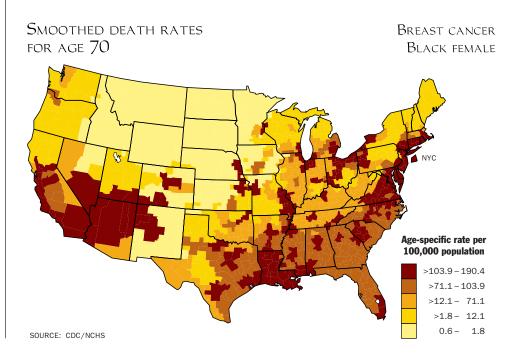


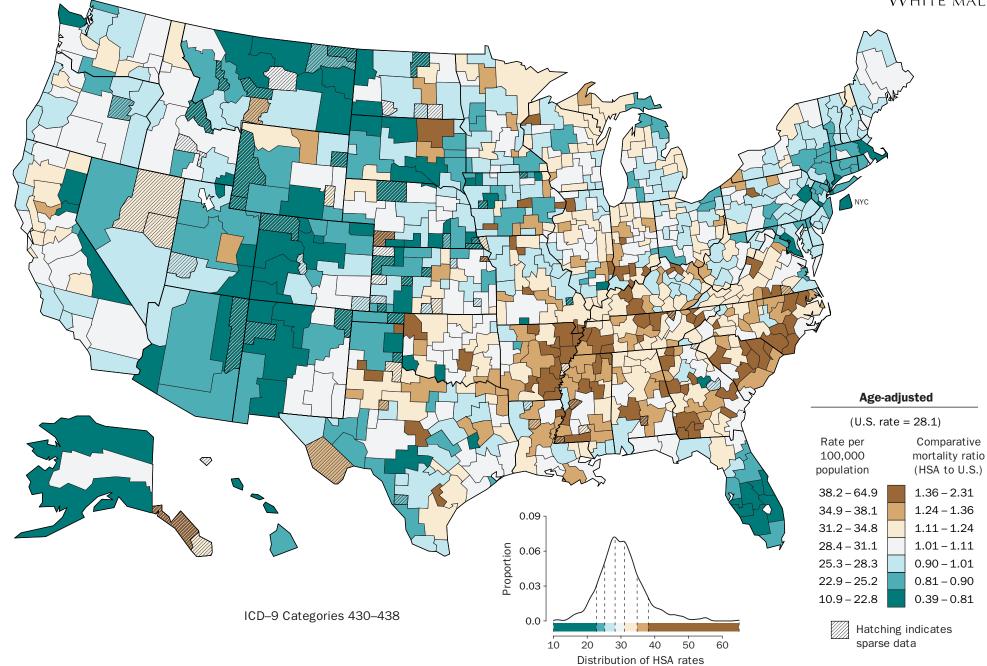


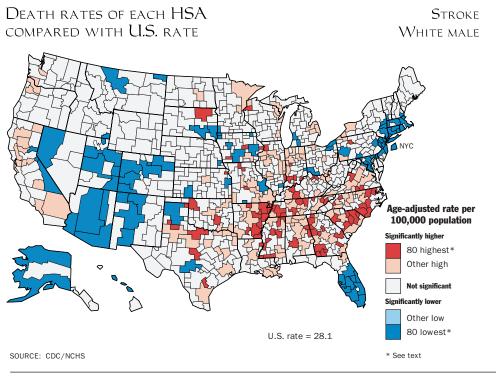
Breast cancer Black female





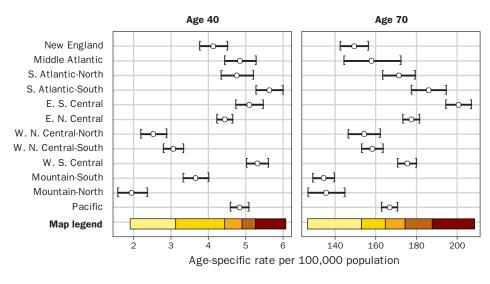


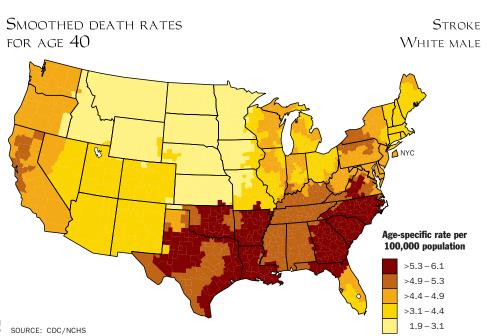


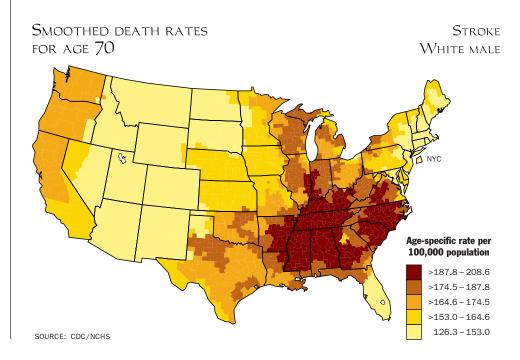


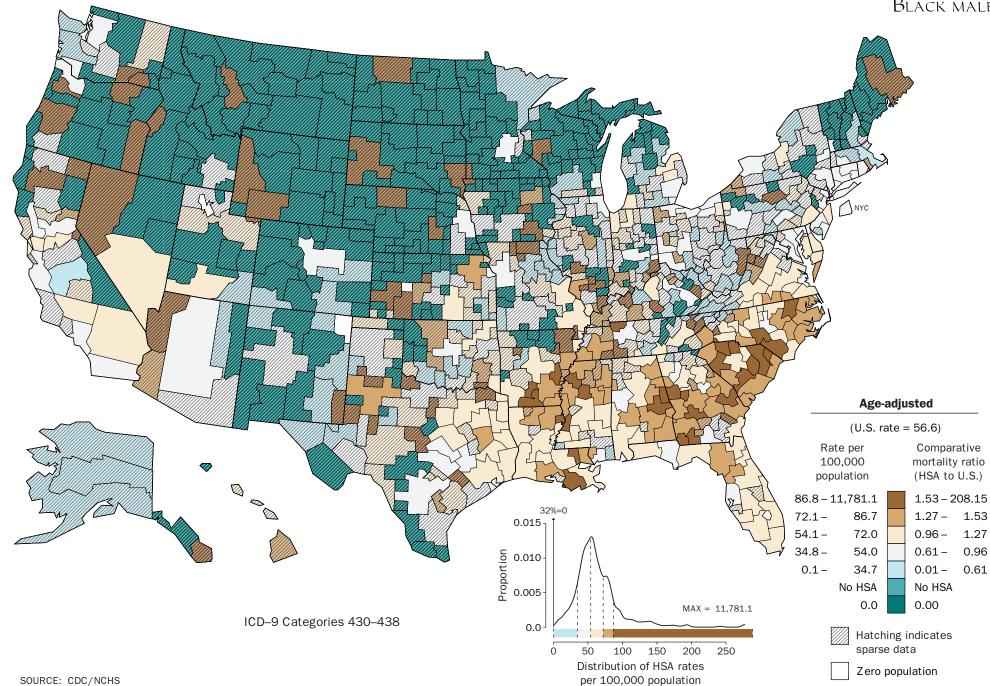


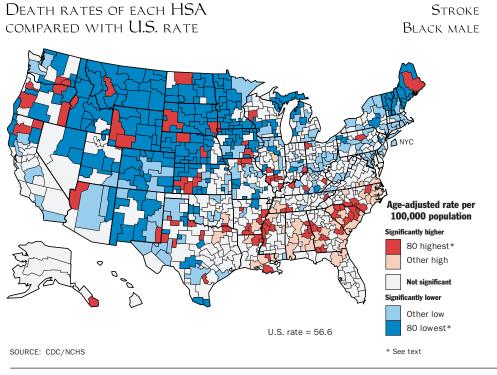






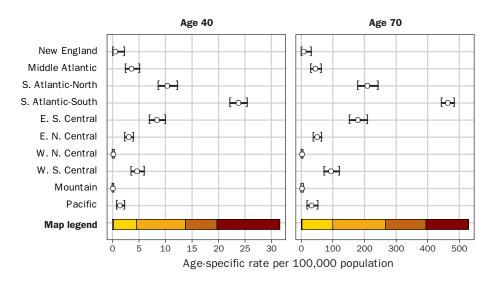


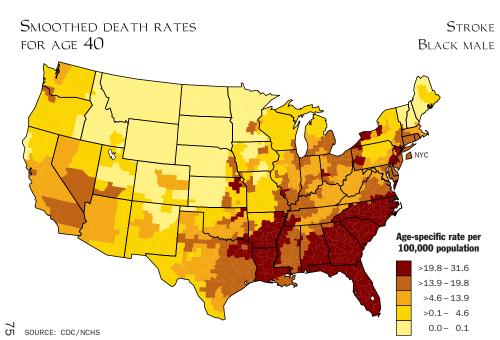


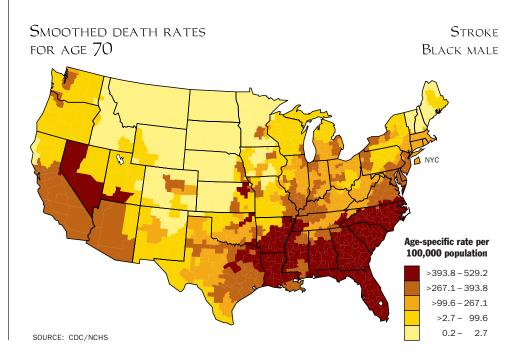


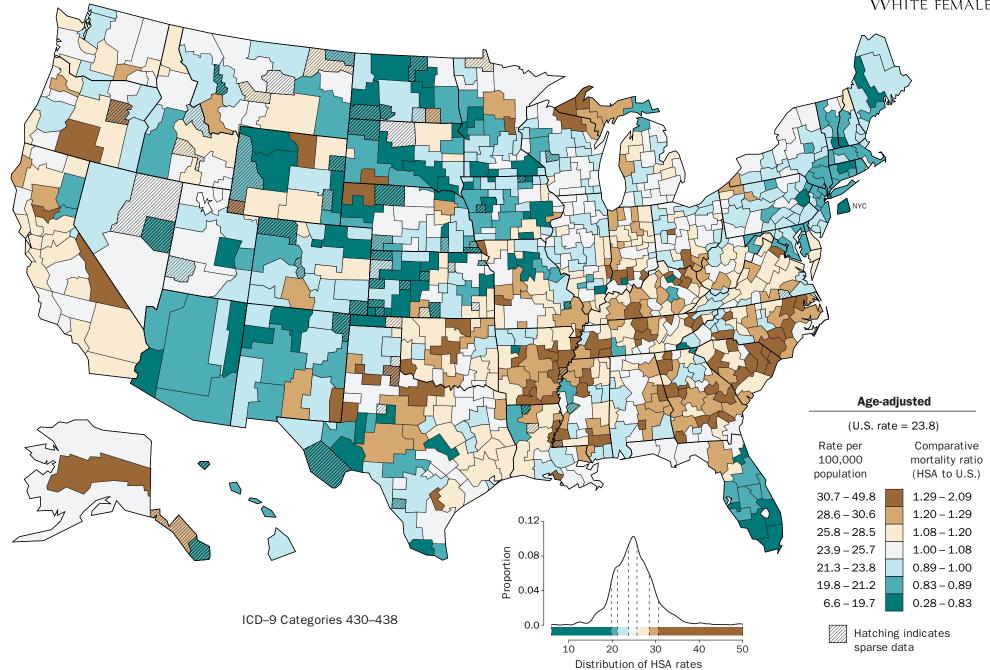


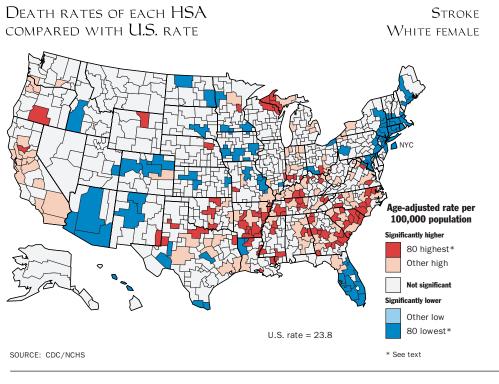






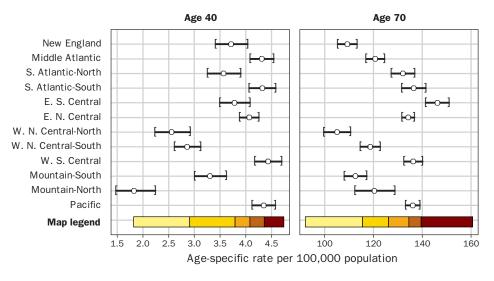


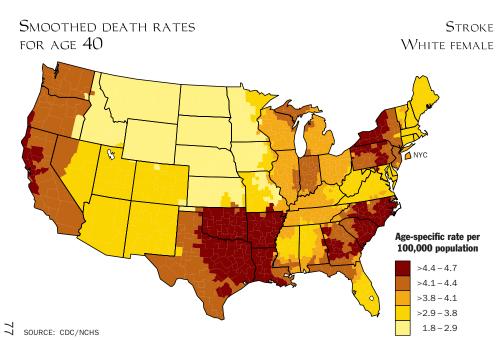


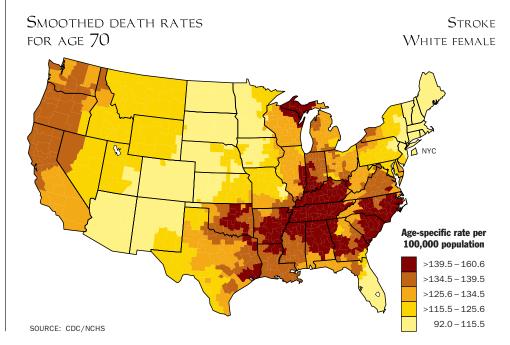


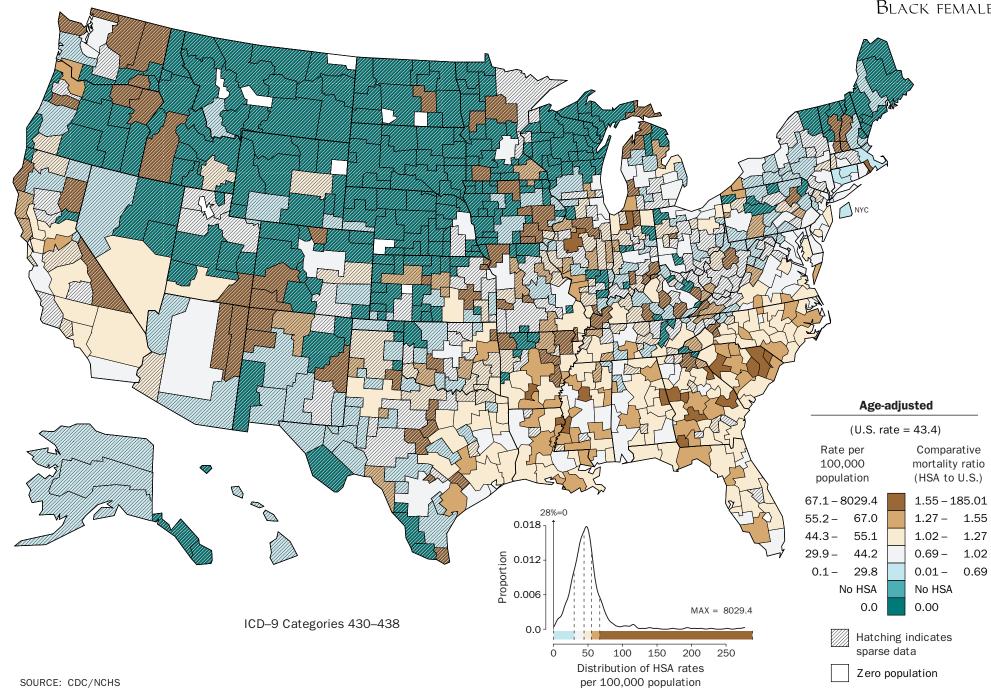


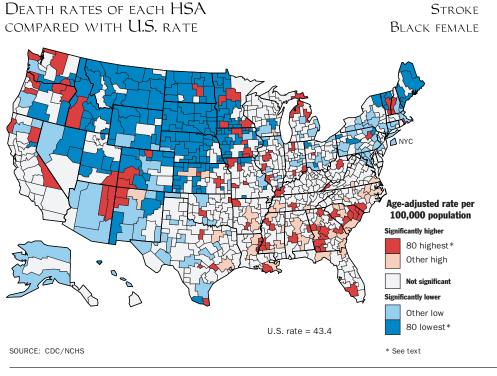
Stroke White female





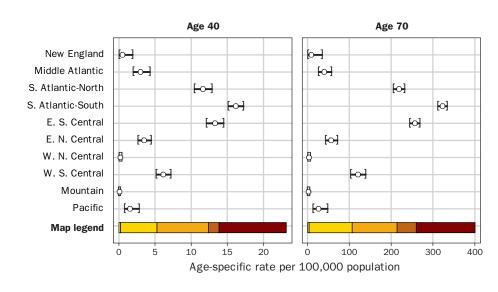


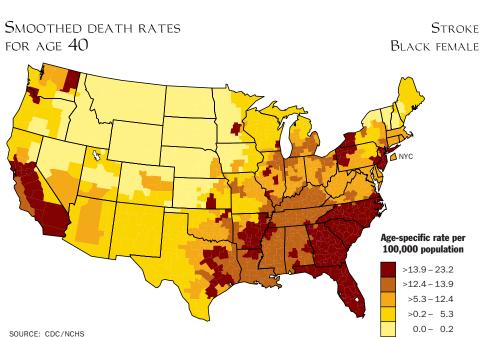


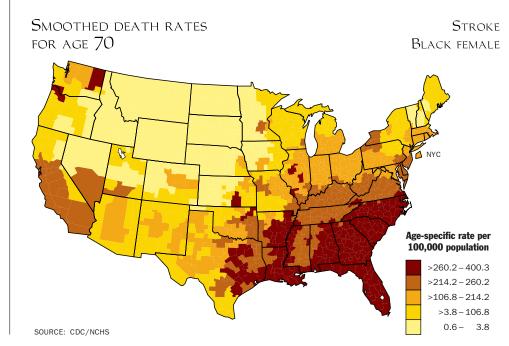


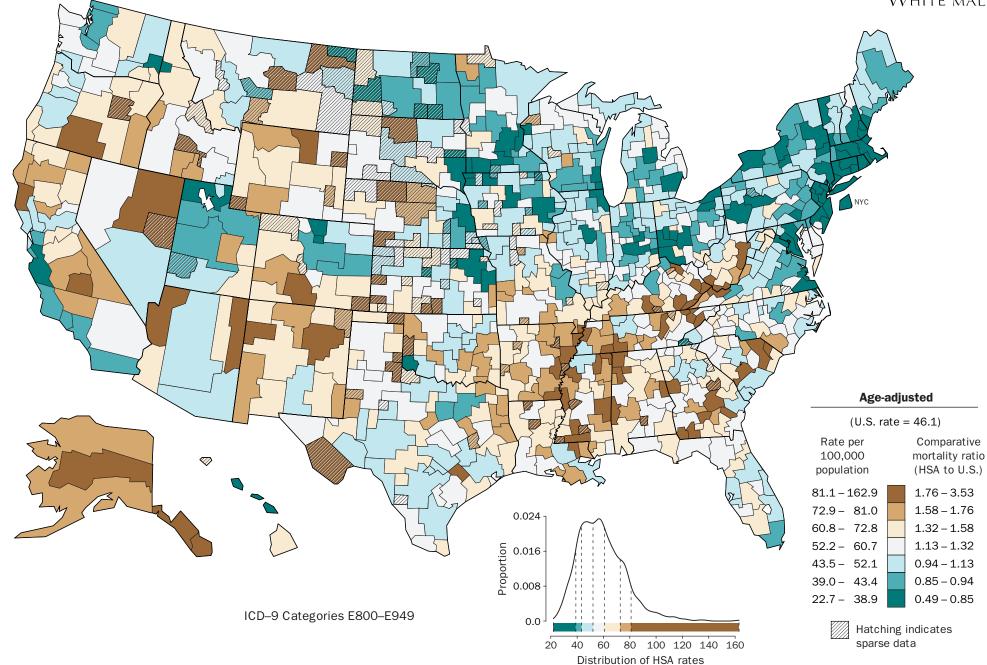


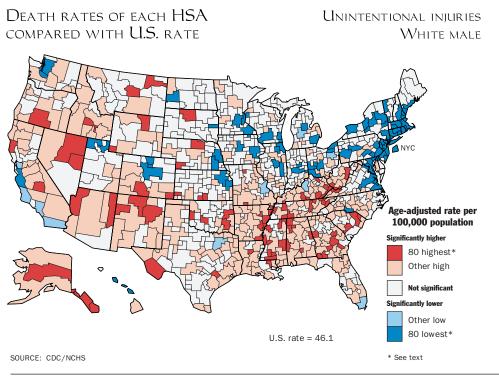






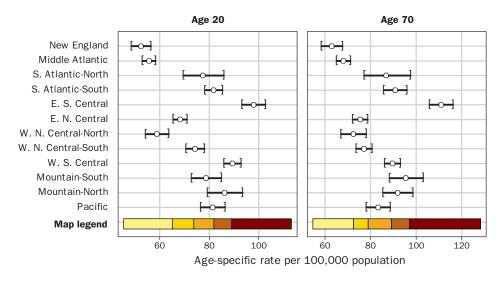


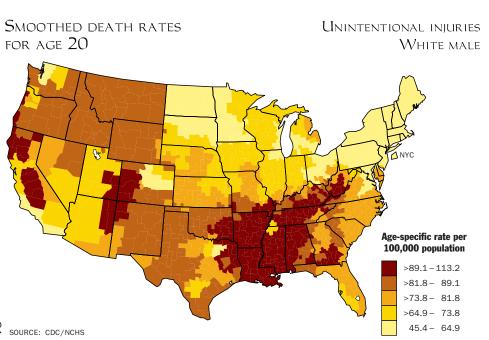


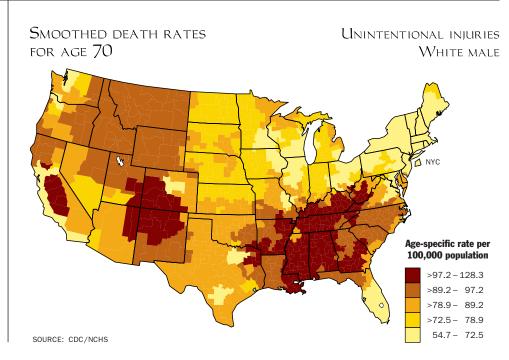


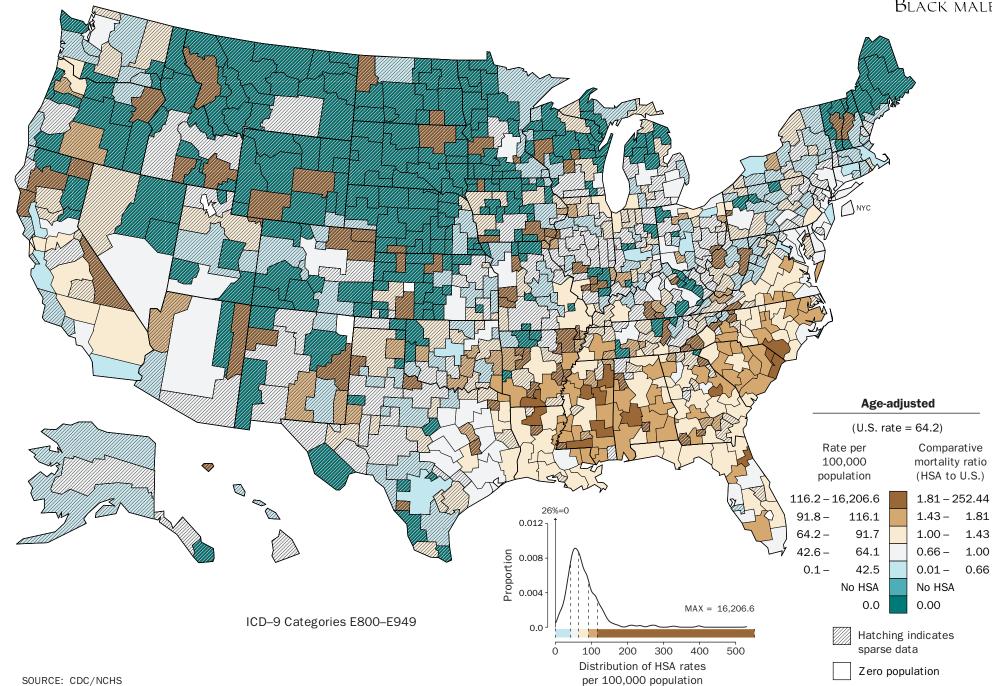
PREDICTED REGIONAL RATES FOR SMOOTHED RATE MAPS

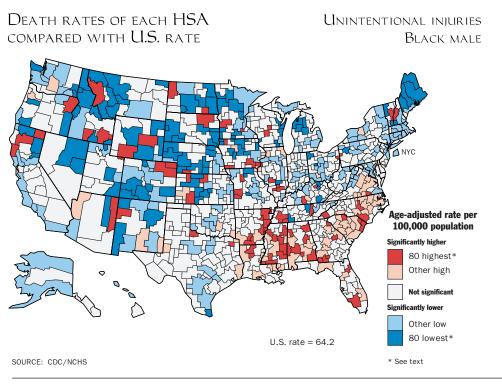
Unintentional injuries
White male





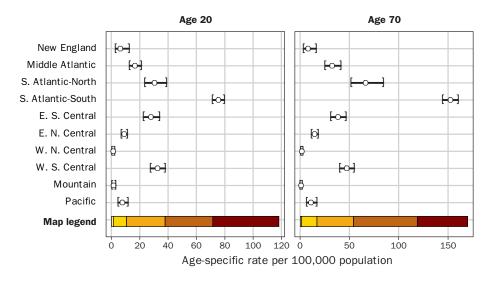


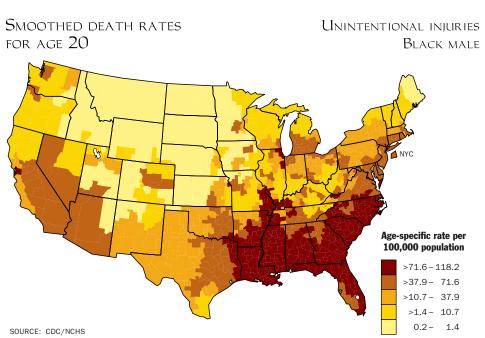


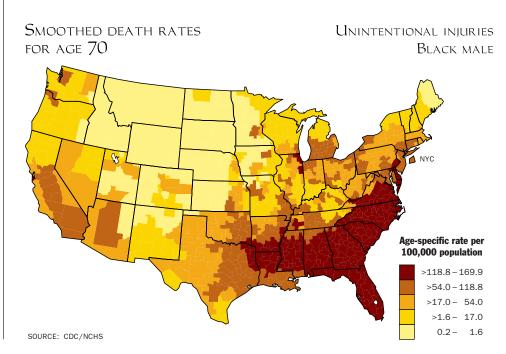


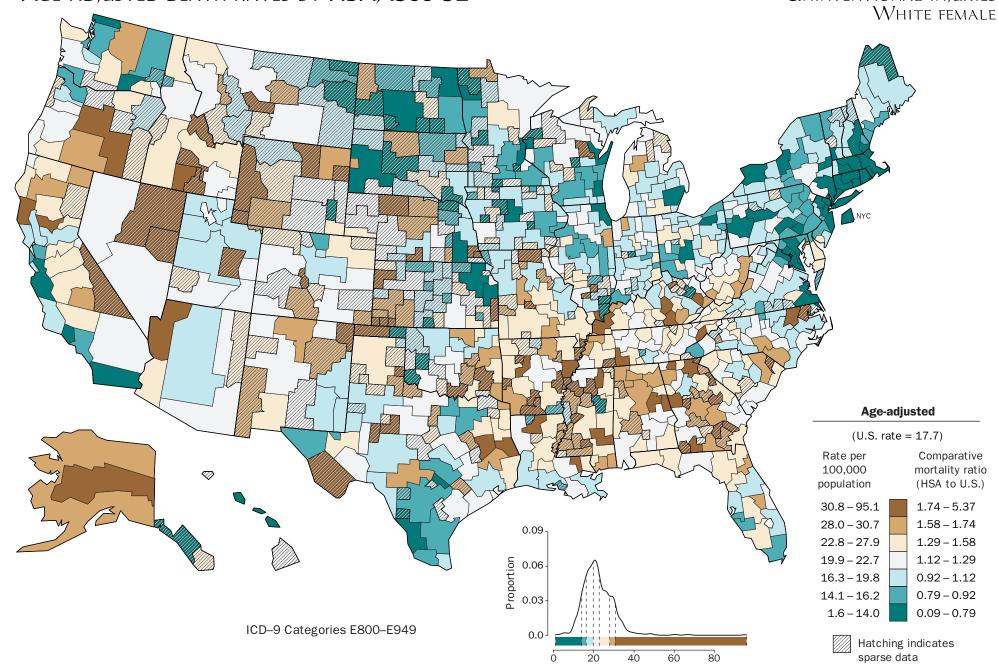
PREDICTED REGIONAL RATES FOR SMOOTHED RATE MAPS

Unintentional injuries
Black male



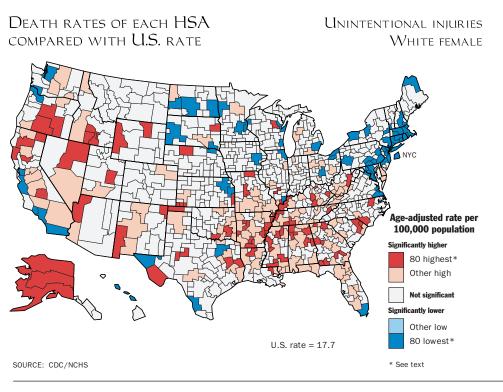






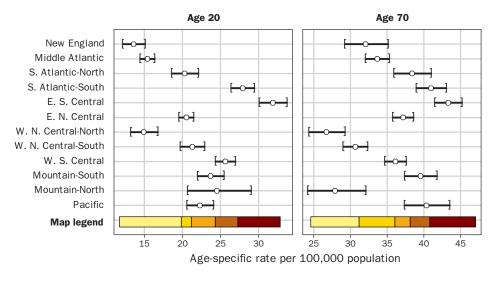
Distribution of HSA rates per 100,000 population

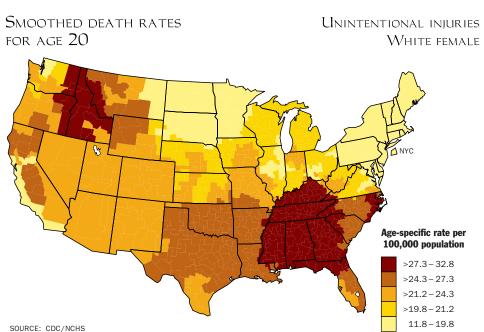
SOURCE: CDC/NCHS

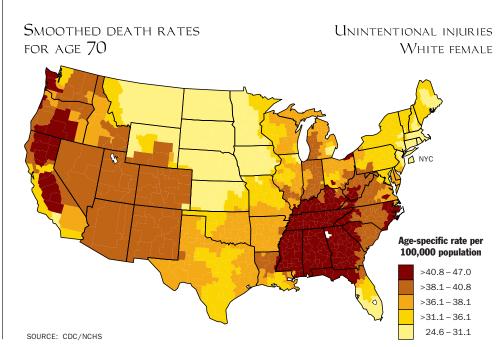


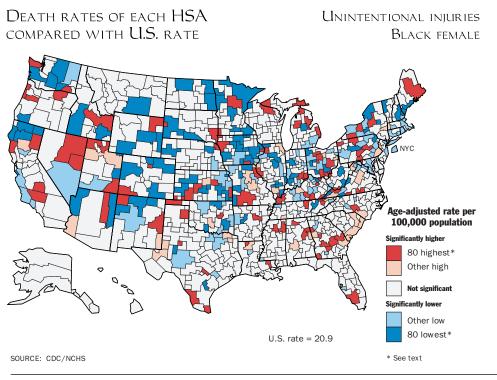
PREDICTED REGIONAL RATES FOR SMOOTHED RATE MAPS

Unintentional injuries
White female



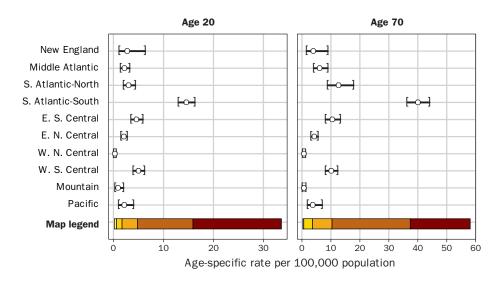


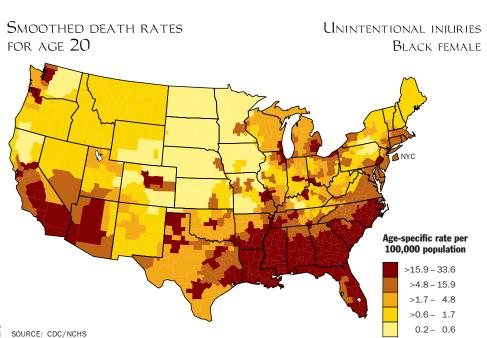


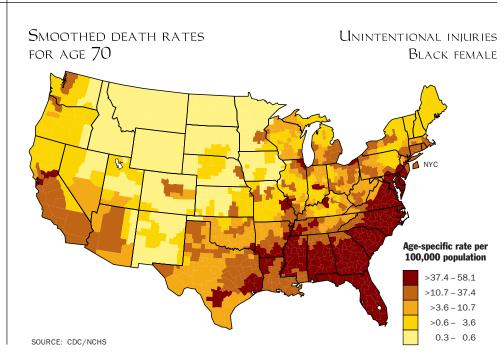


PREDICTED REGIONAL RATES FOR SMOOTHED RATE MAPS

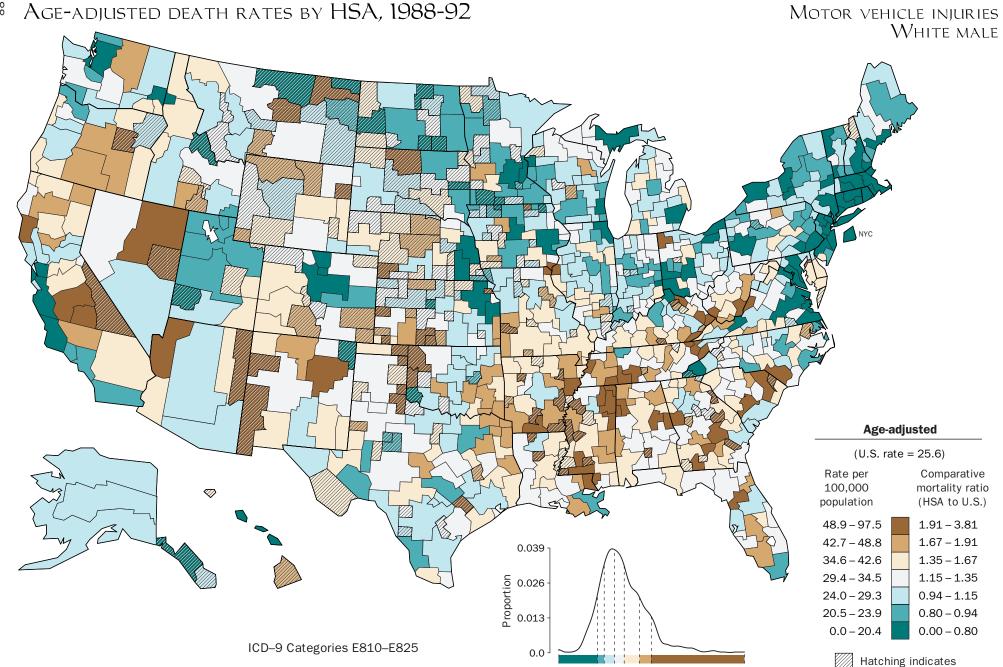
Unintentional injuries
Black female







sparse data



20

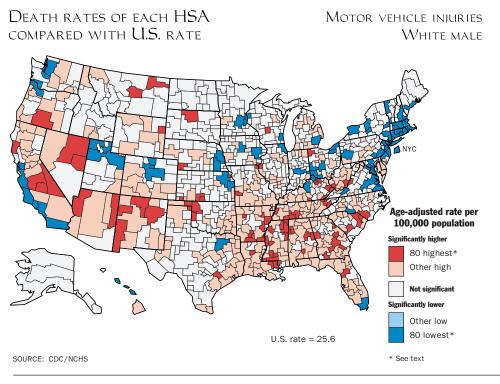
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Distribution of HSA rates per 100,000 population

60

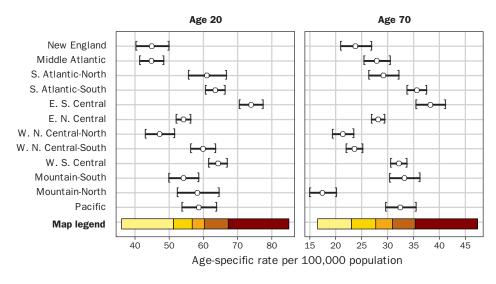
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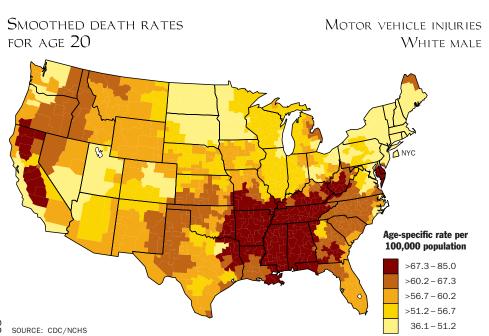
SOURCE: CDC/NCHS

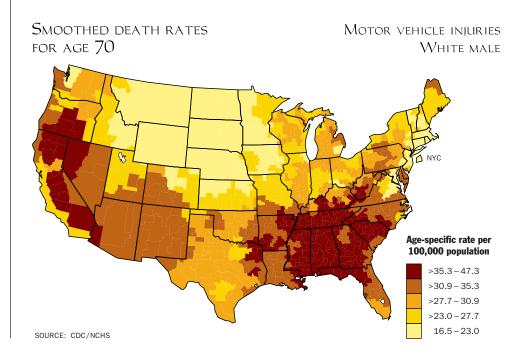


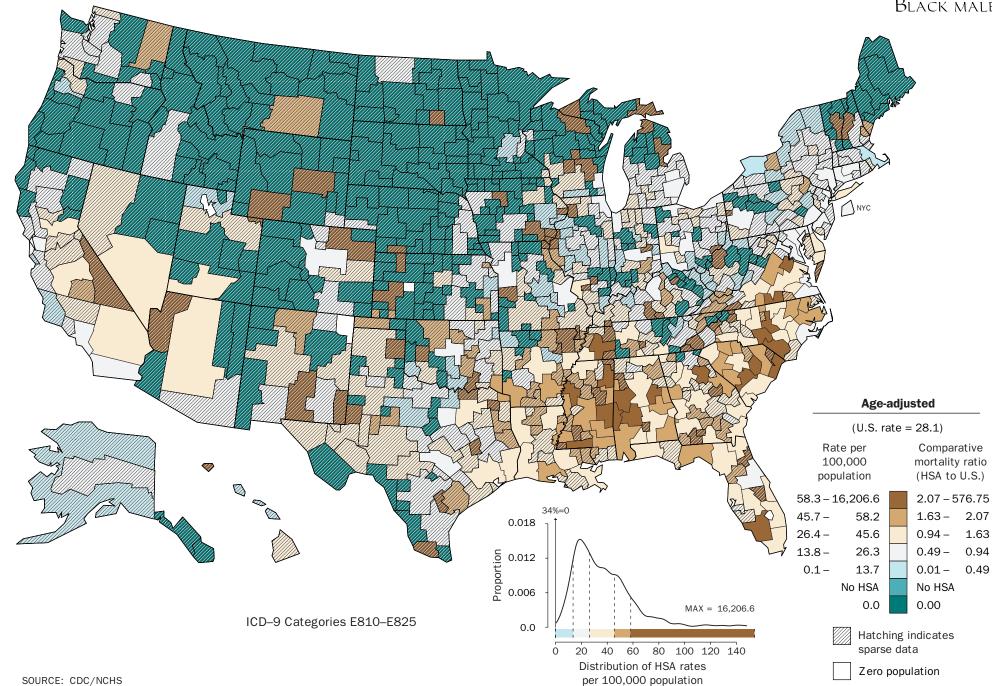
PREDICTED REGIONAL RATES FOR SMOOTHED RATE MAPS

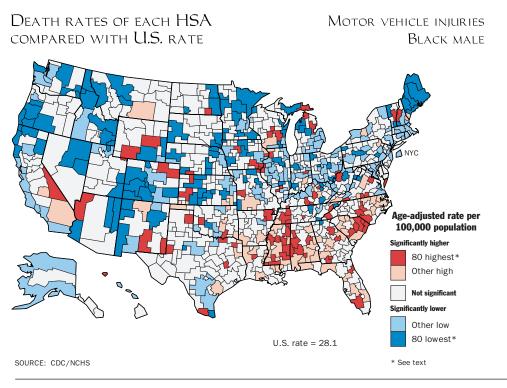
Motor vehicle injuries
White male





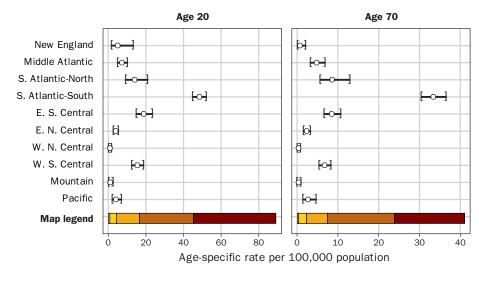


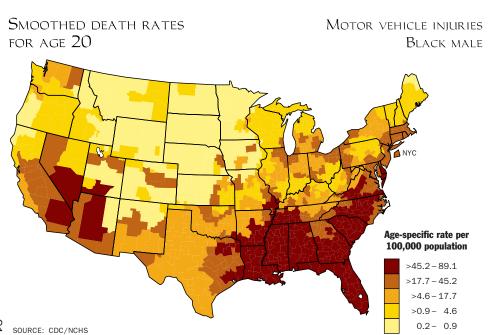


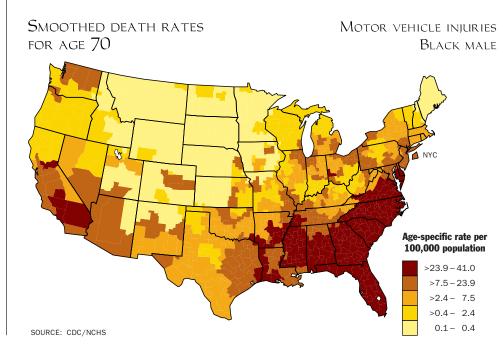


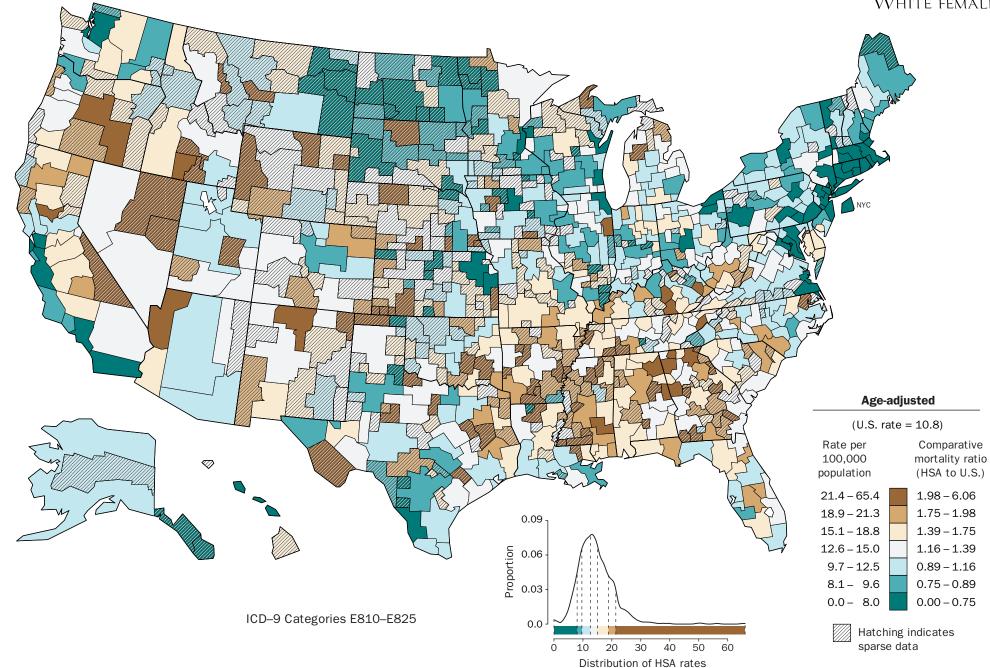
Predicted regional rates FOR SMOOTHED RATE MAPS

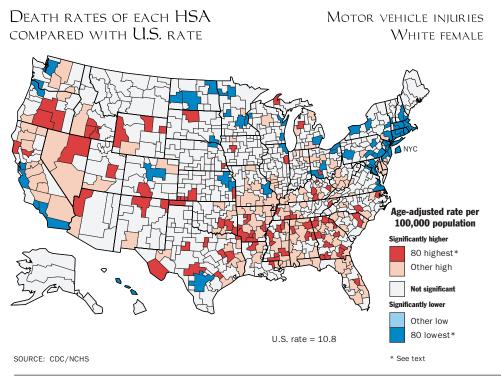
Motor vehicle injuries BLACK MALE





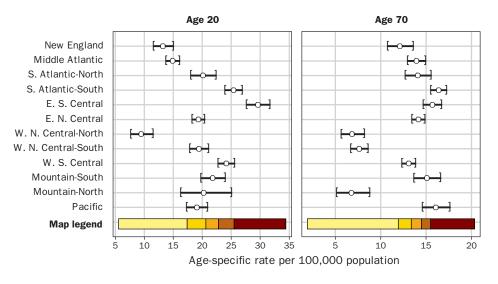


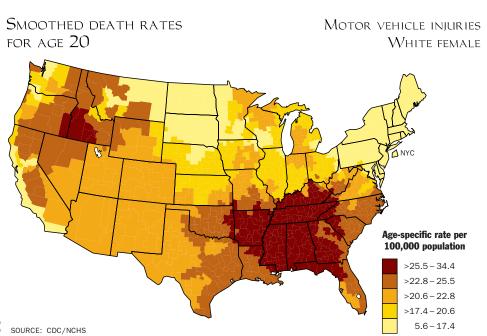


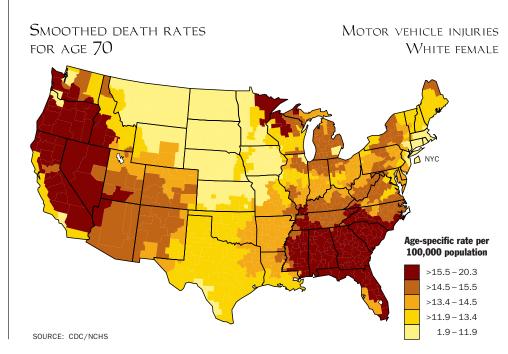


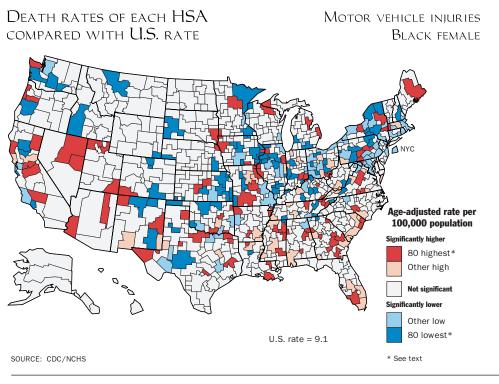
Predicted regional rates FOR SMOOTHED RATE MAPS

MOTOR VEHICLE INJURIES White female



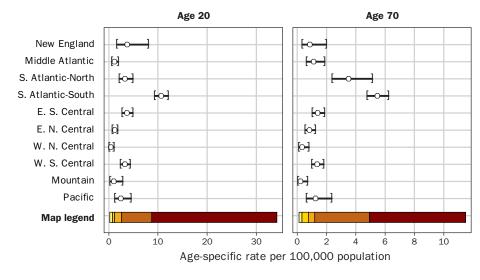


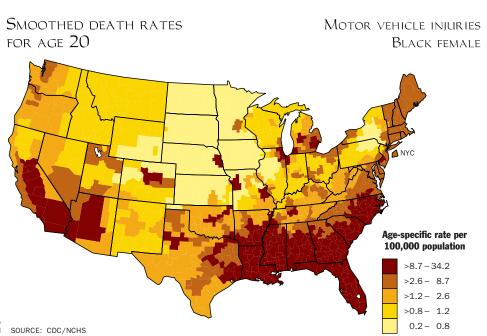


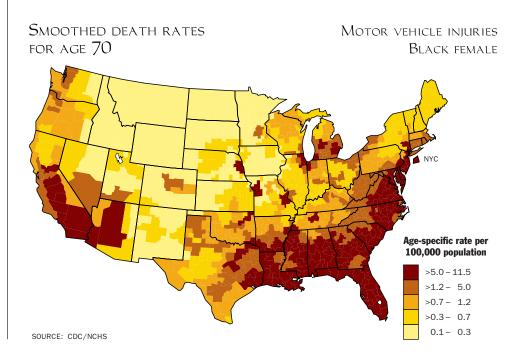


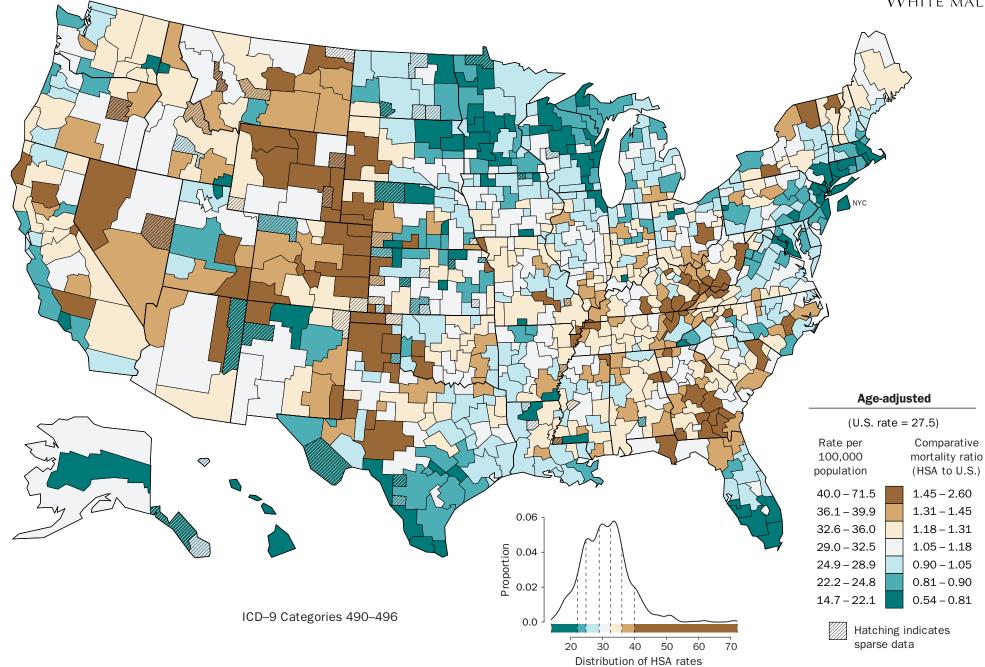
Predicted regional rates for smoothed rate maps

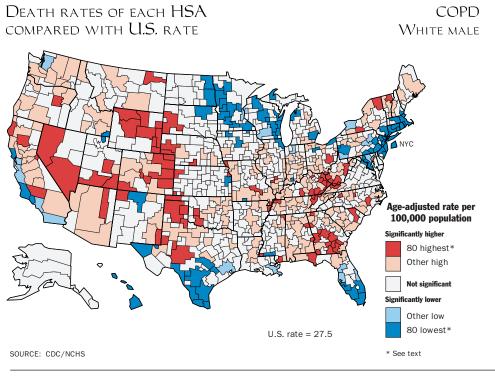
MOTOR VEHICLE INJURIES
BLACK FEMALE





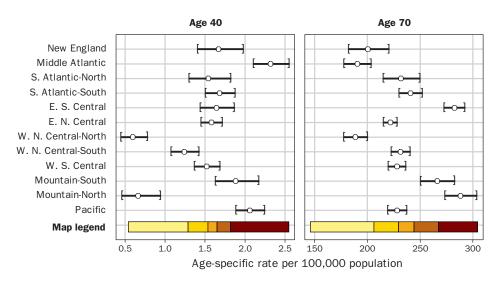


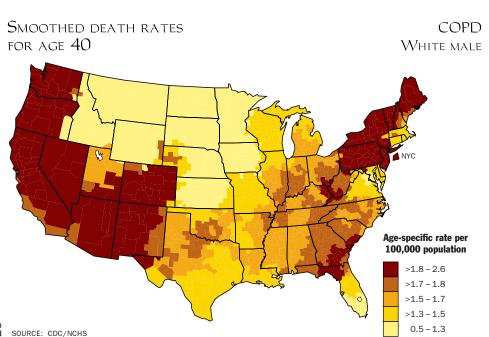


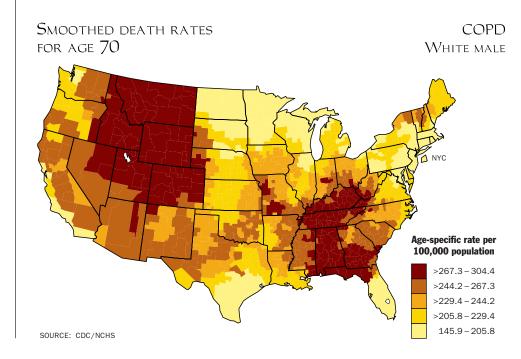


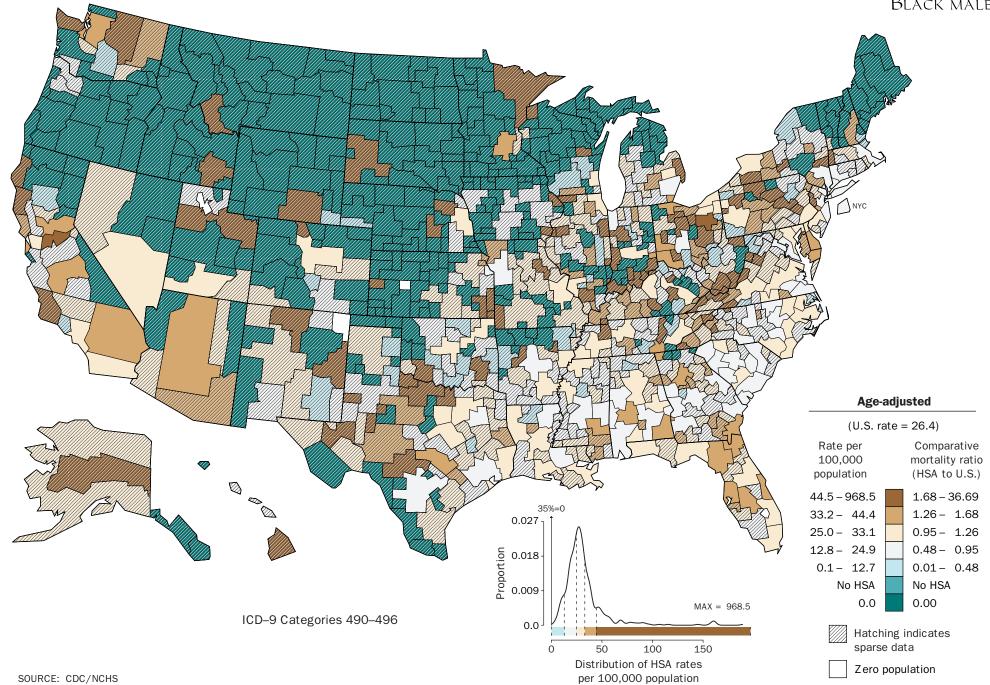


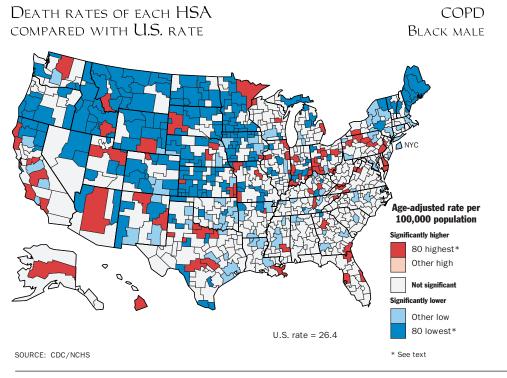






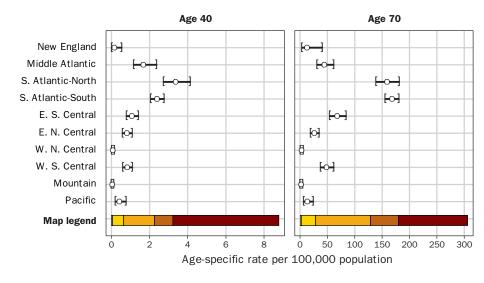


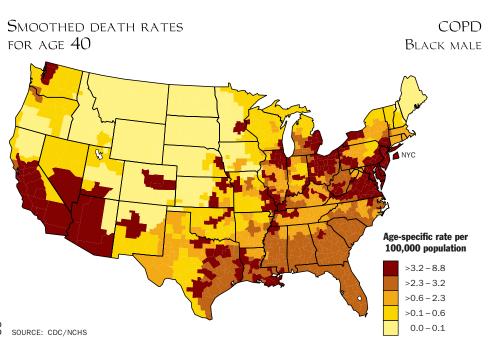


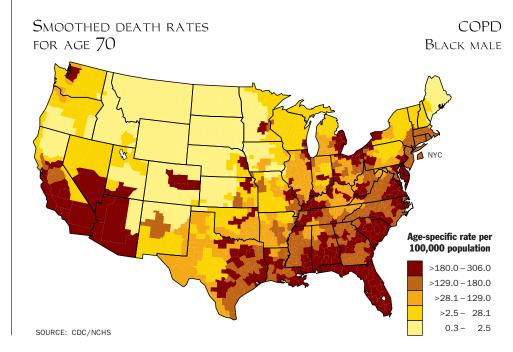




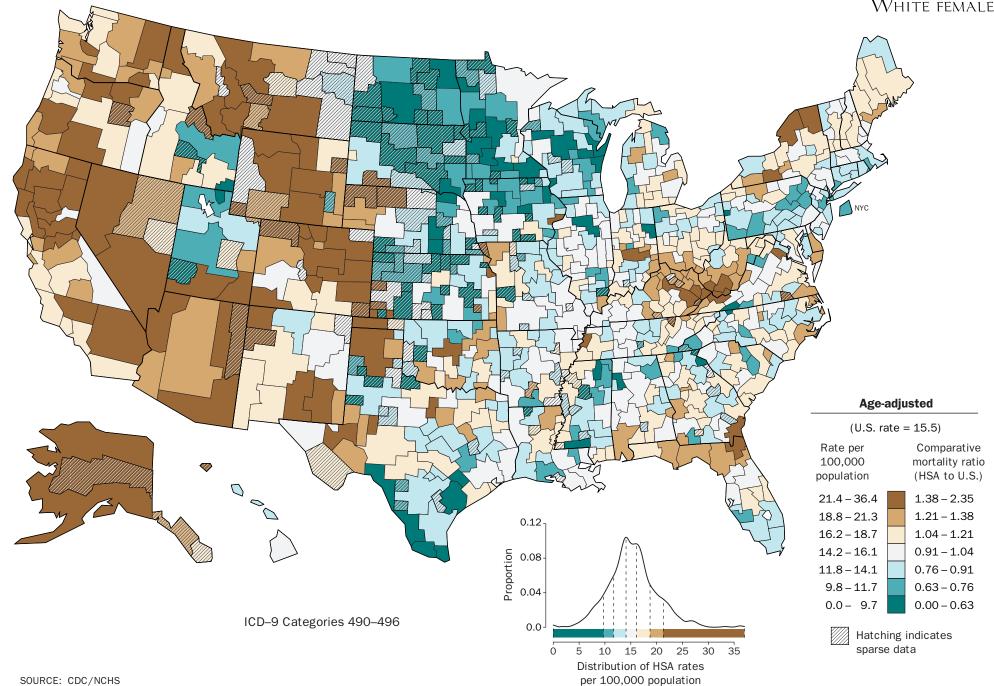


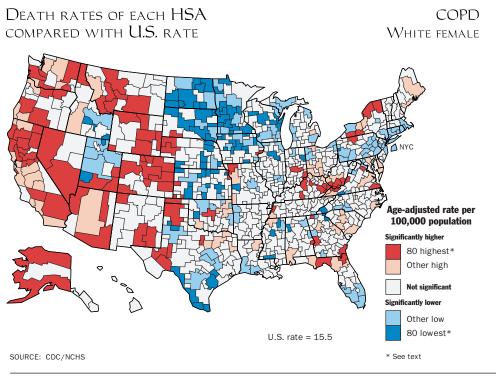






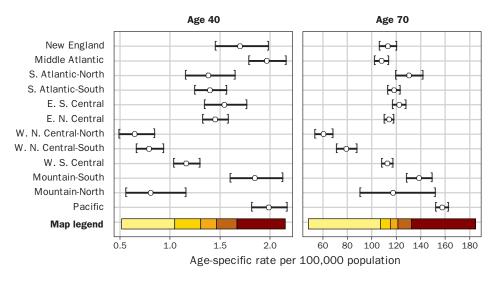


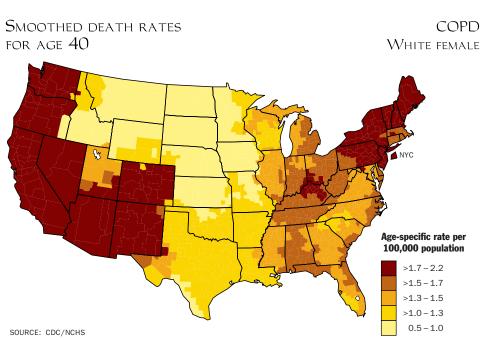


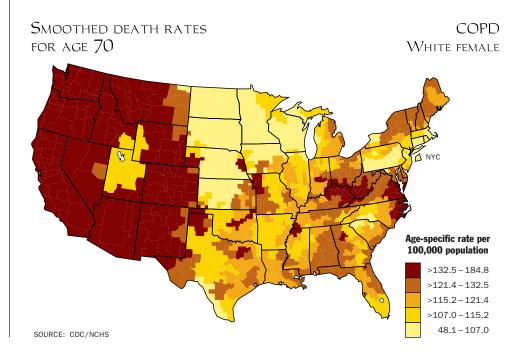


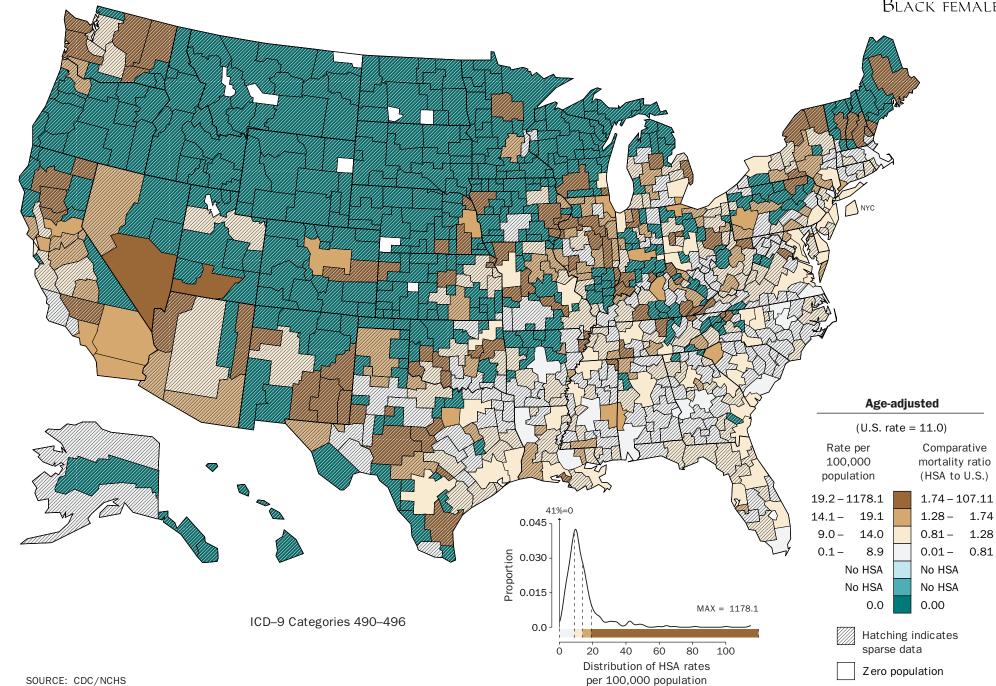


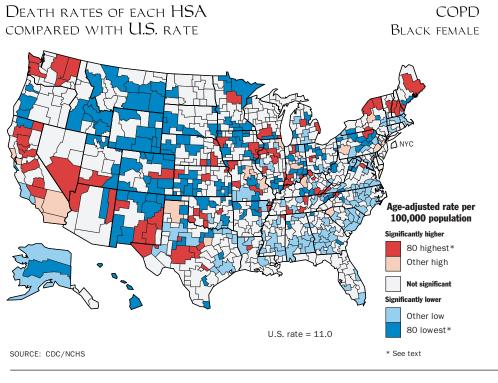






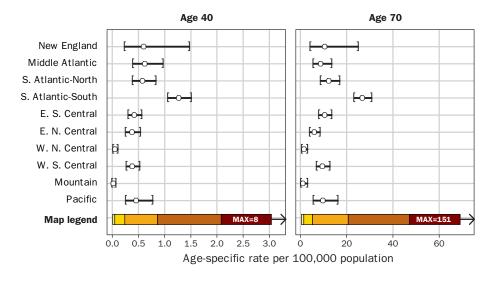


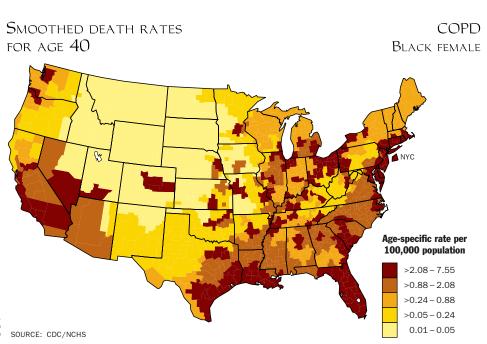


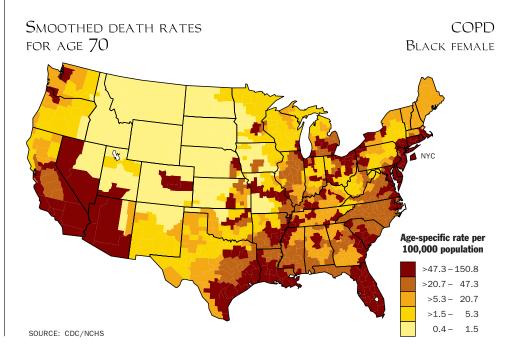


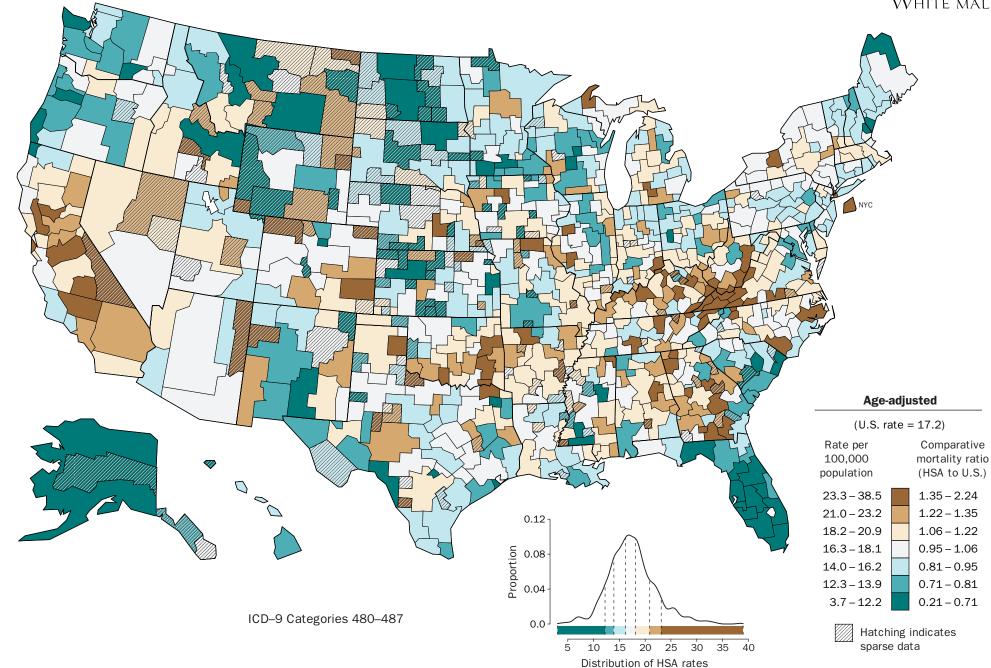


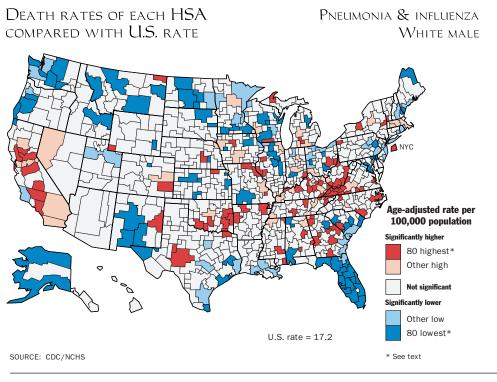






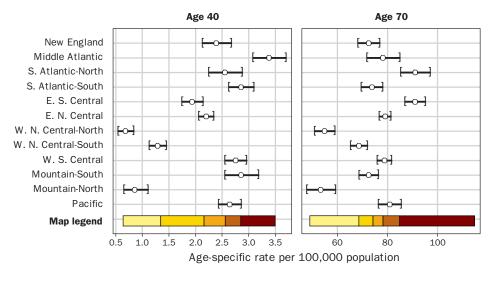


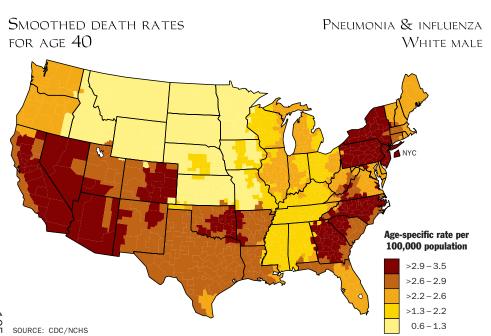


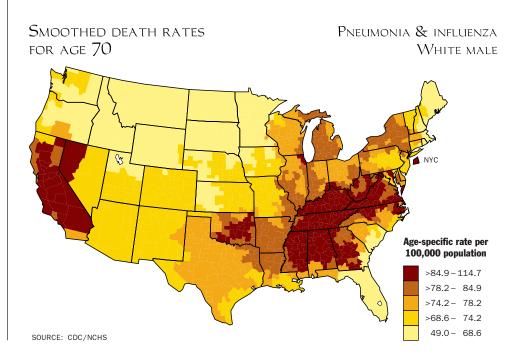




Pneumonia & influenza White male

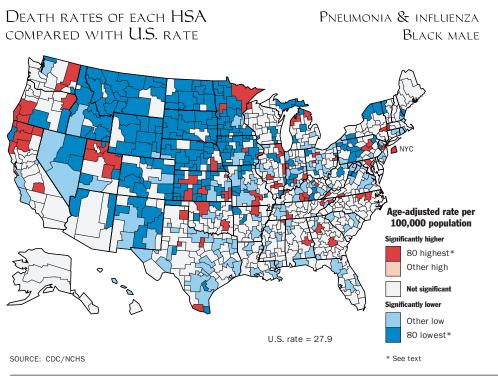






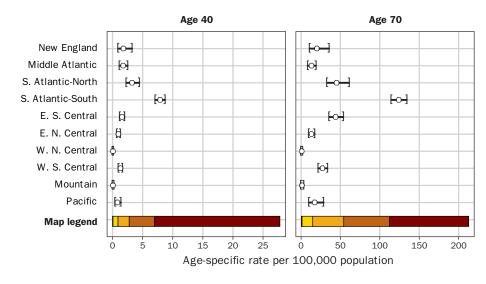
Zero population

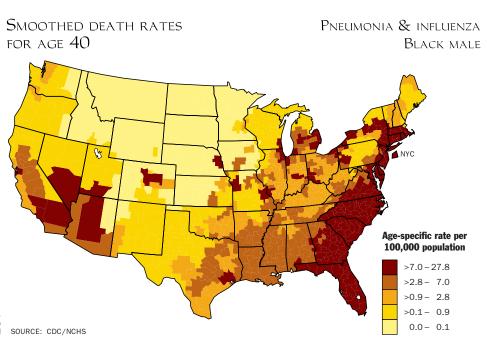
Distribution of HSA rates

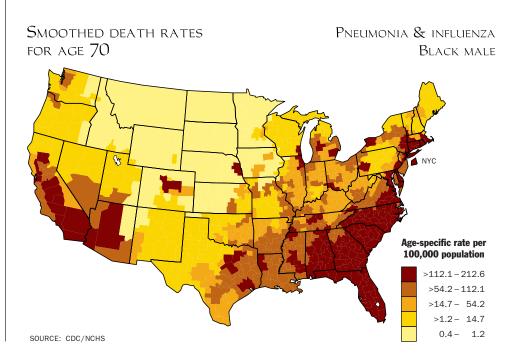


PREDICTED REGIONAL RATES FOR SMOOTHED RATE MAPS

PNEUMONIA & INFLUENZA BLACK MALE





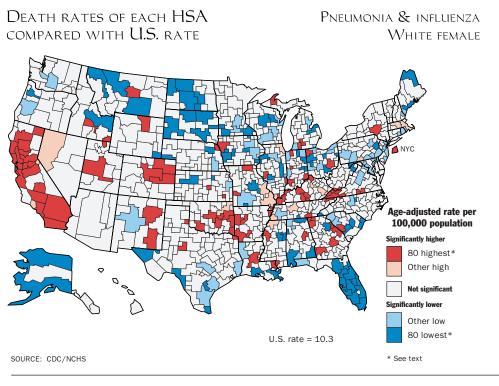


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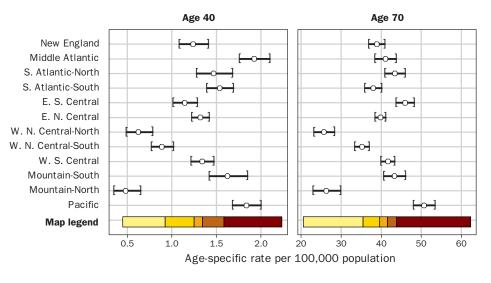
Distribution of HSA rates per 100,000 population

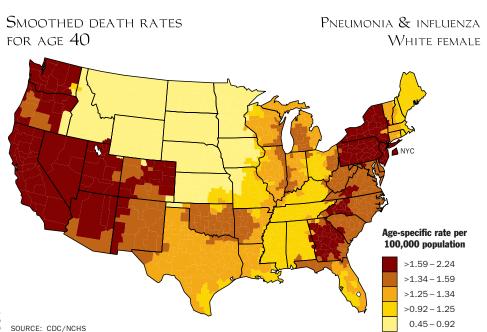
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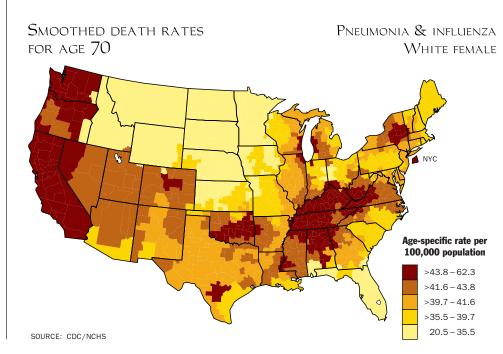


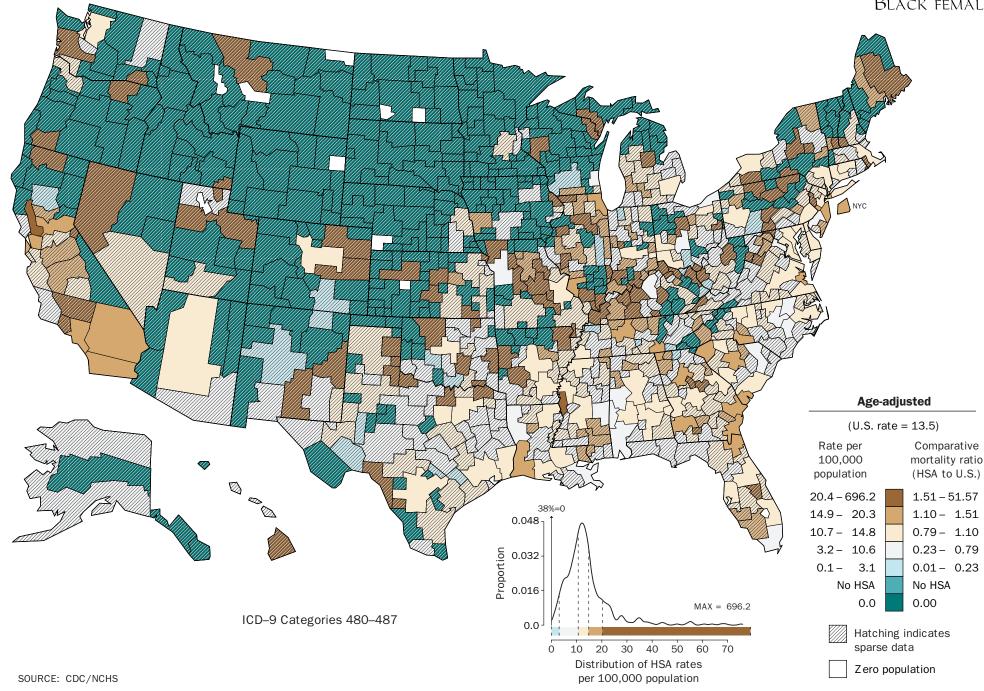


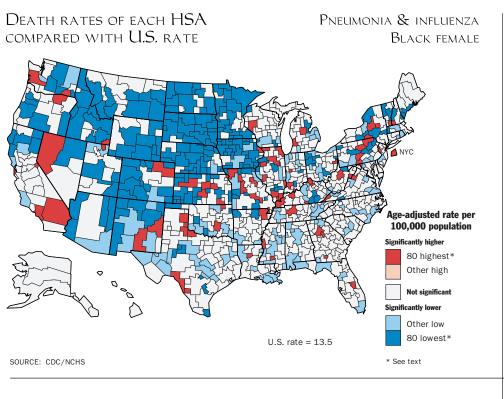
Pneumonia & influenza White female





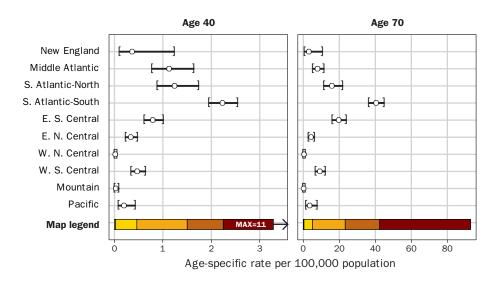


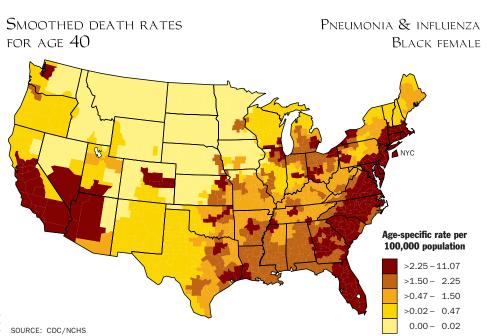


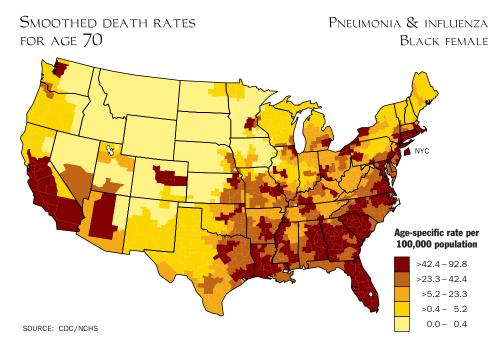


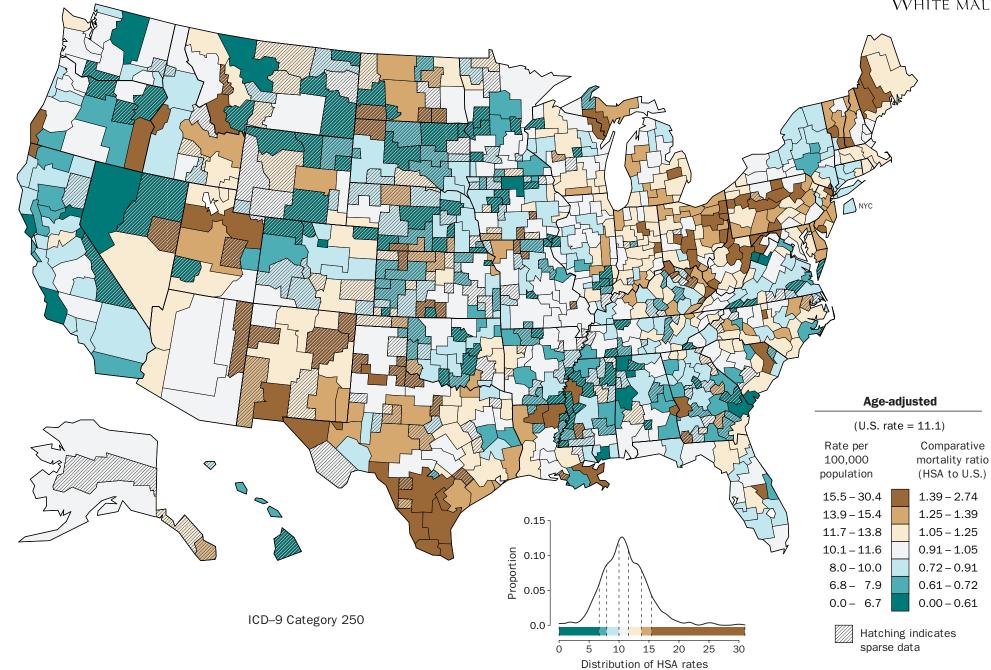


PNEUMONIA & INFLUENZA BLACK FEMALE

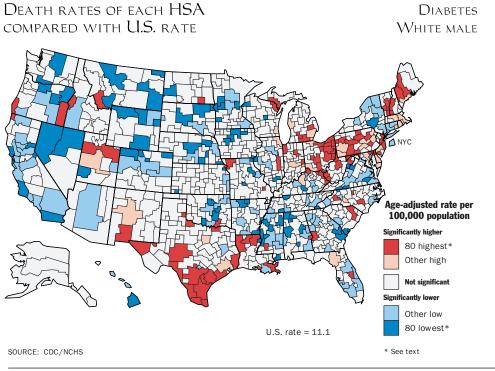




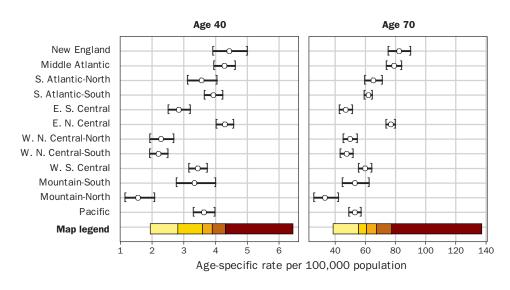


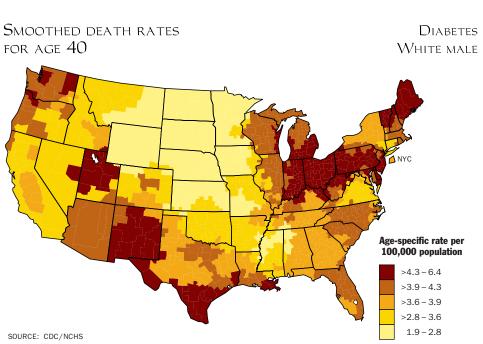


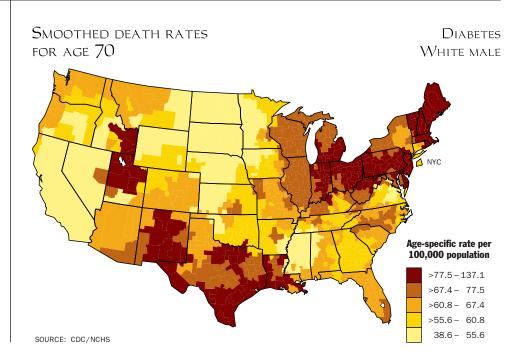
per 100,000 population

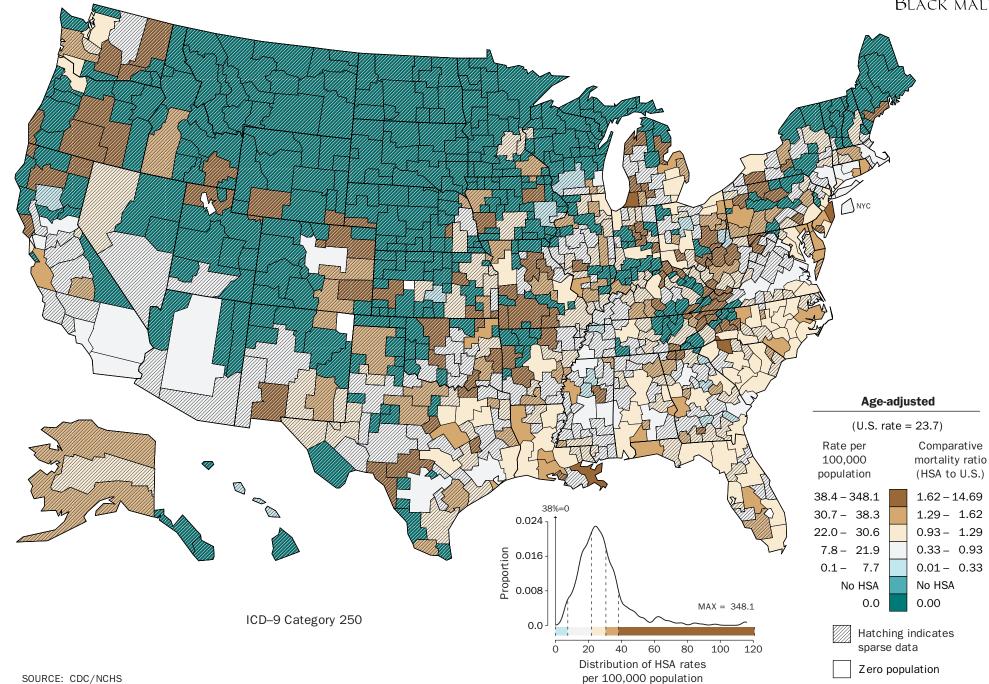


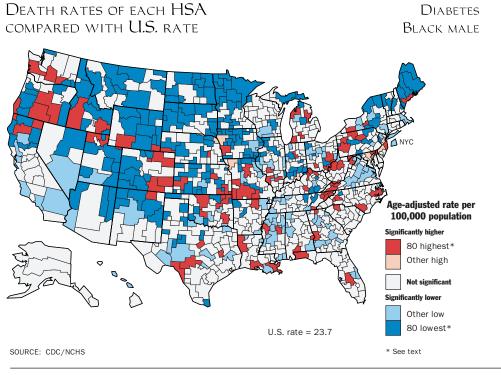
Diabetes White male





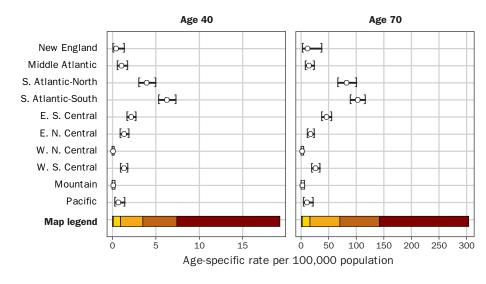


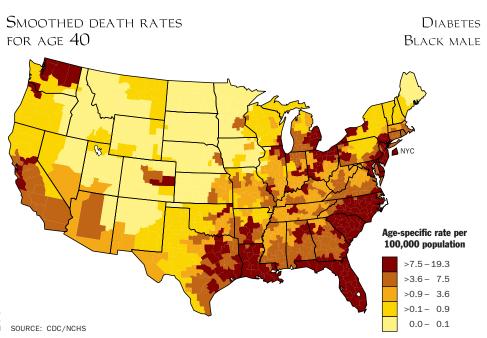


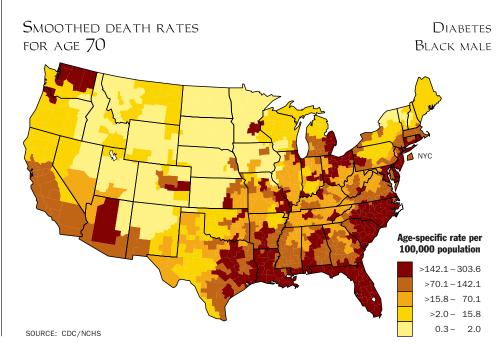




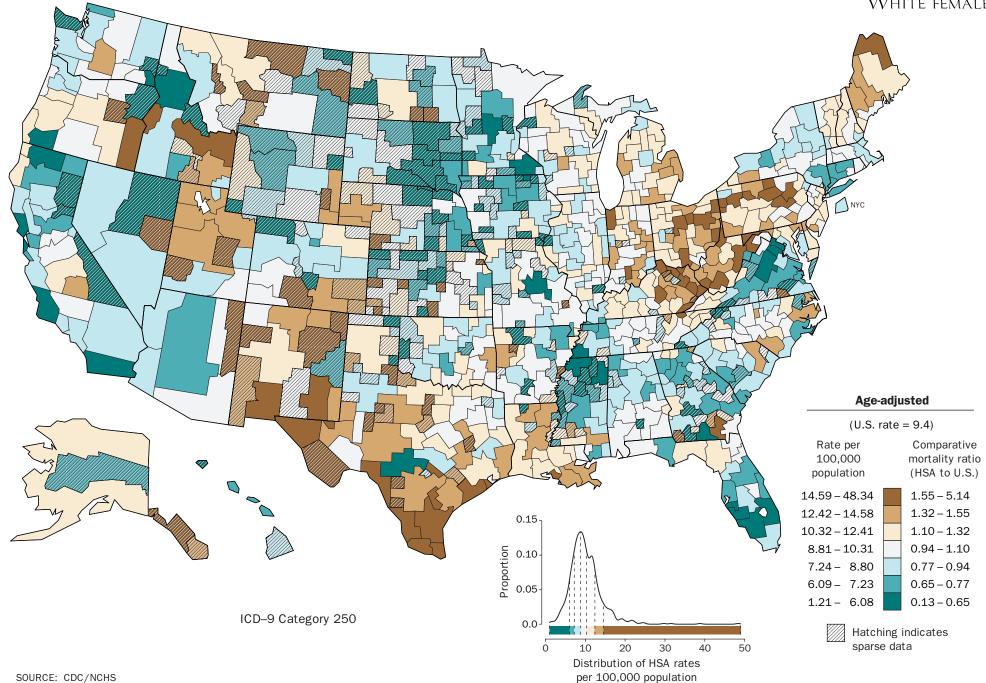
Diabetes Black male

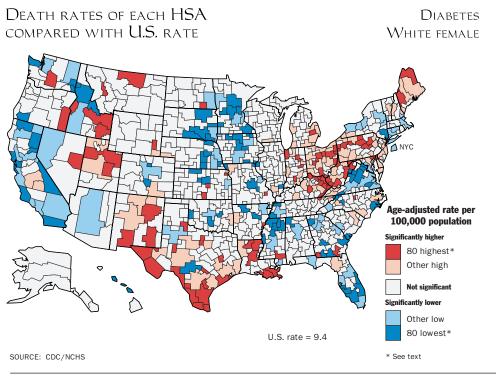






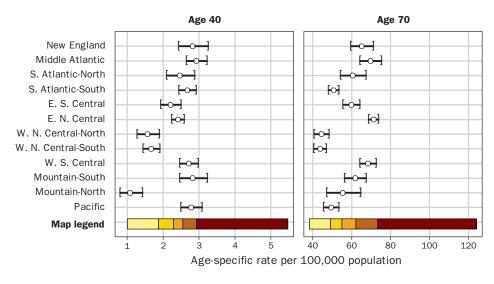


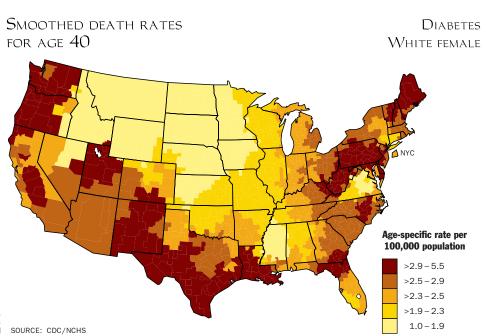


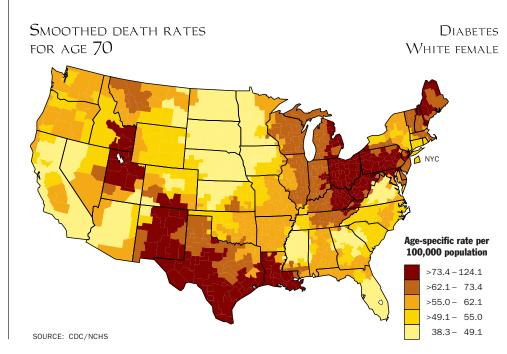


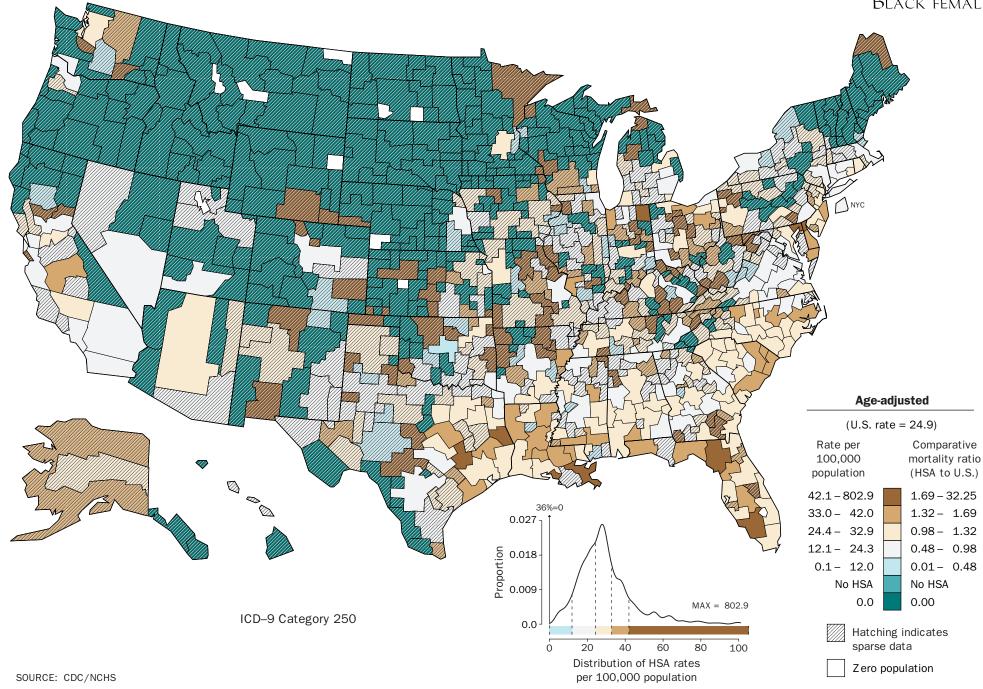


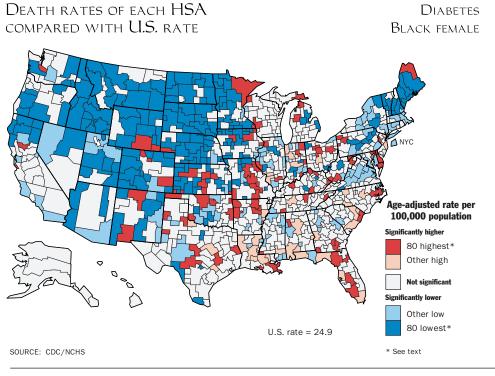






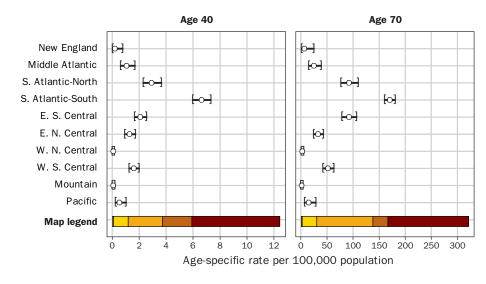


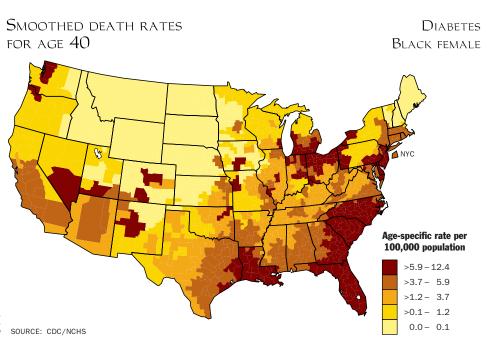


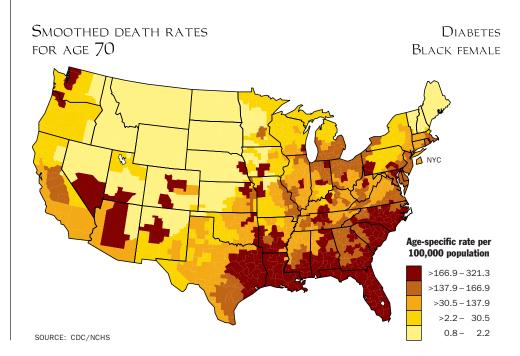


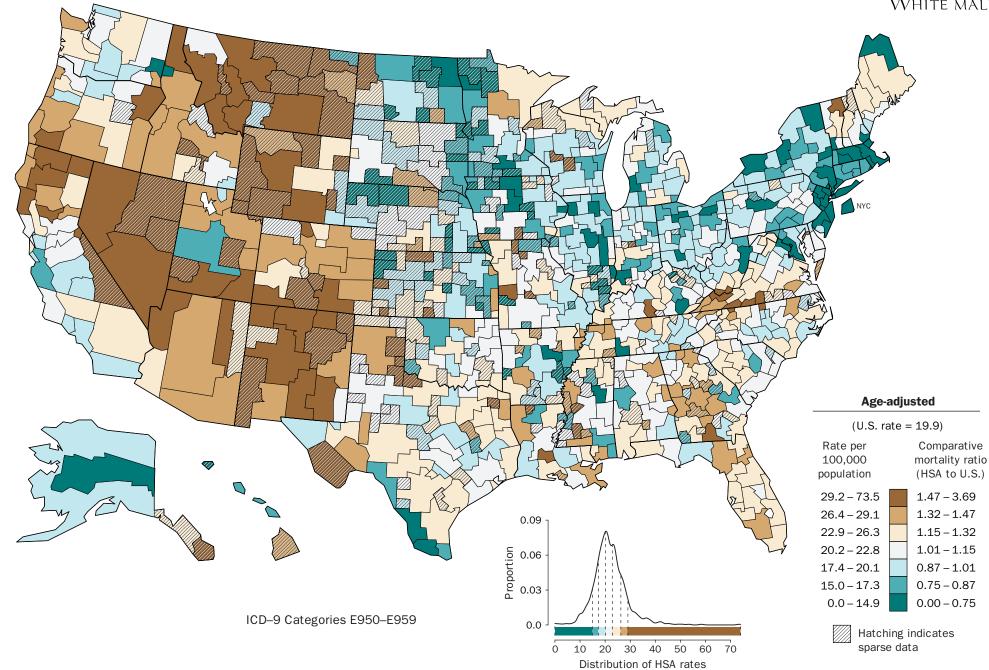


DIABETES BLACK FEMALE

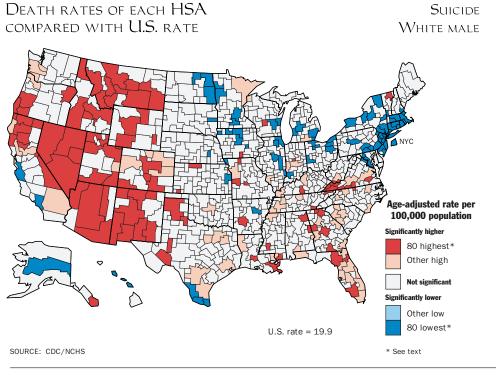






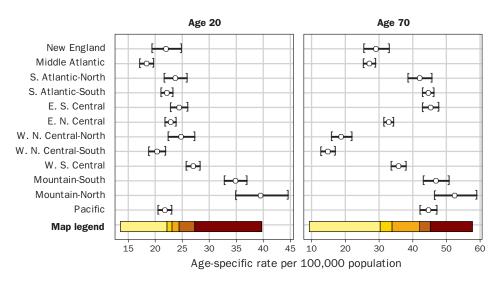


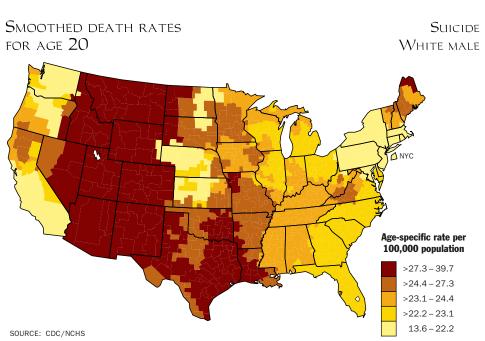
per 100,000 population

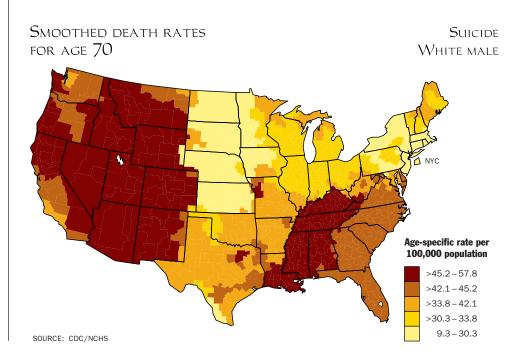


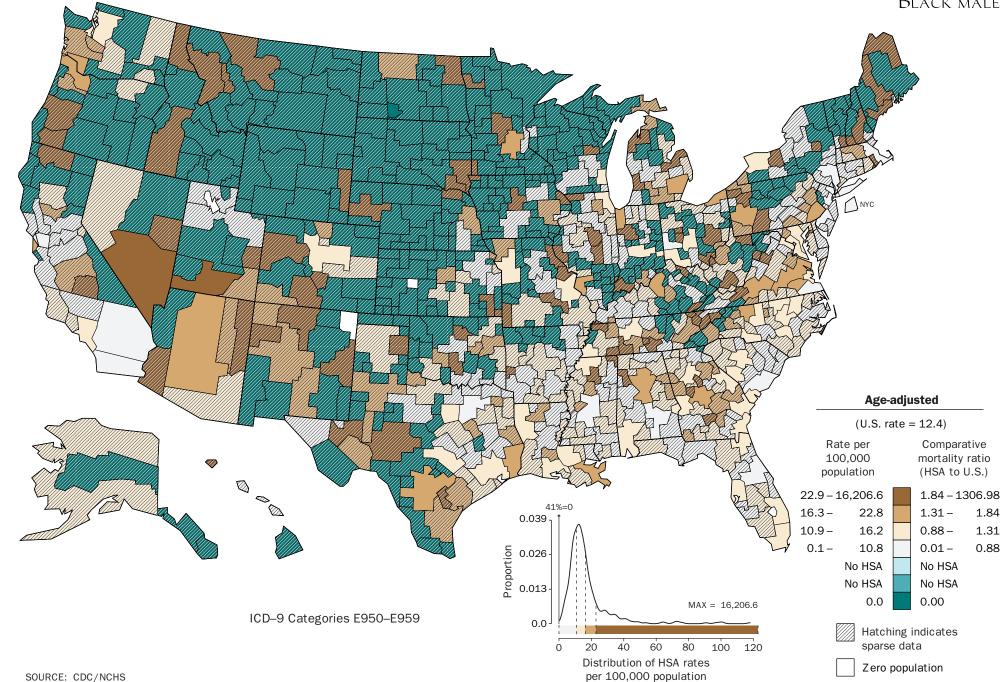
PREDICTED REGIONAL RATES FOR SMOOTHED RATE MAPS

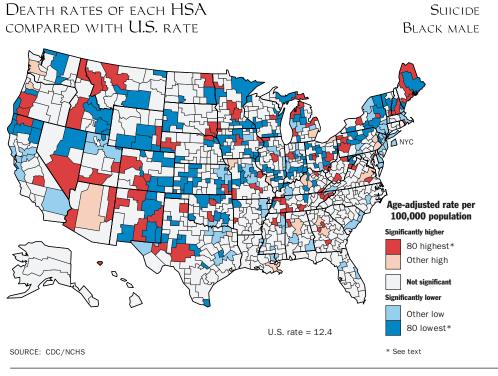
Suicide White male



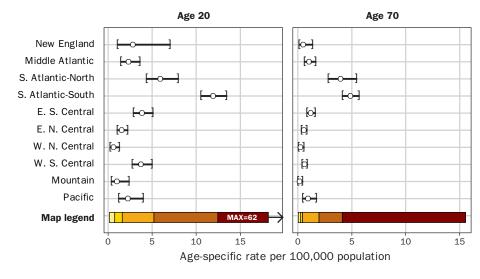


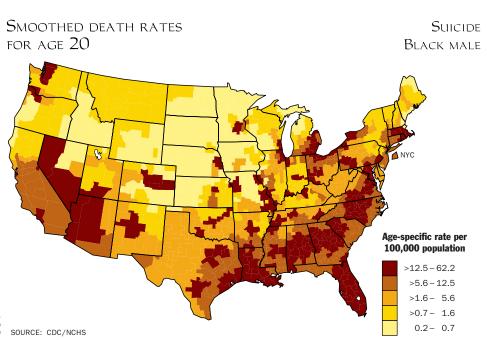


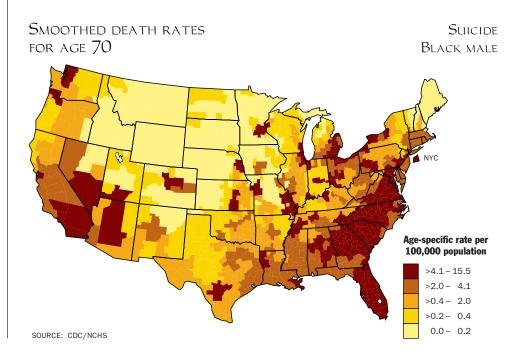


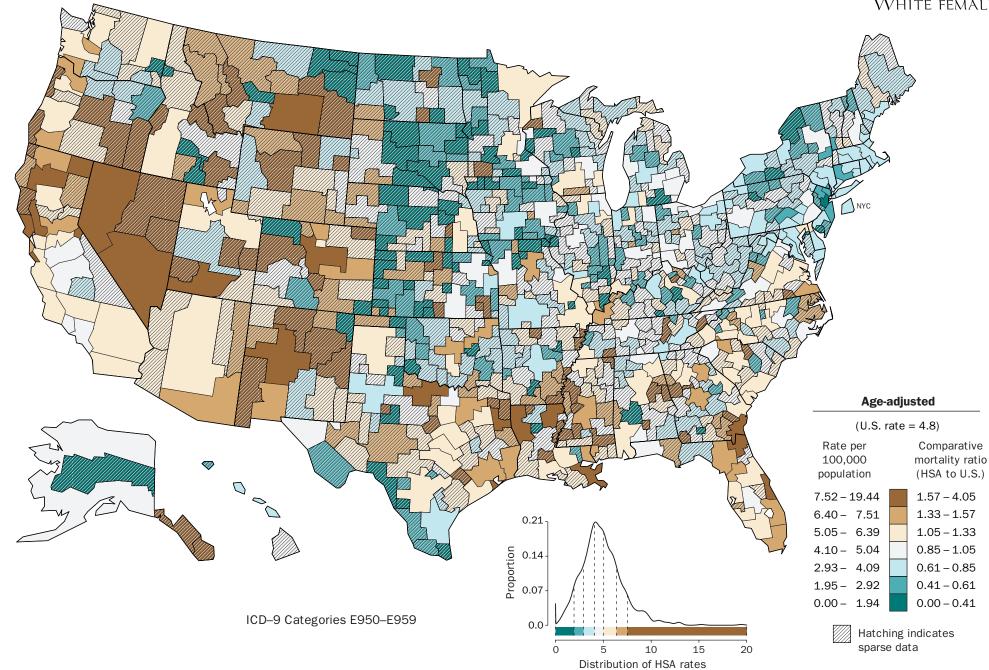


SUICIDE BLACK MALE

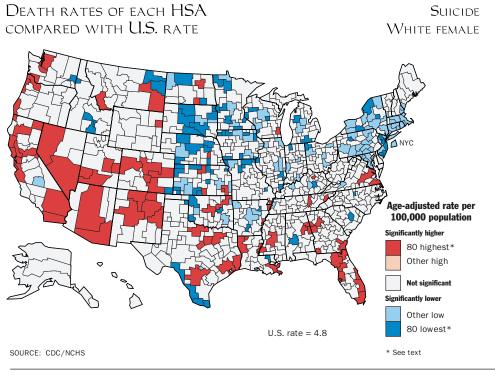






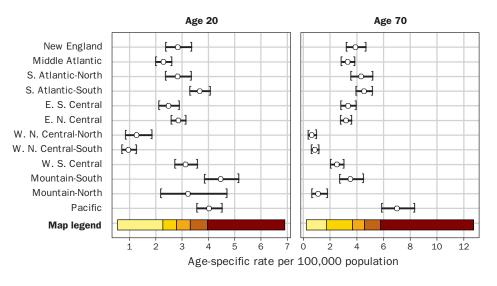


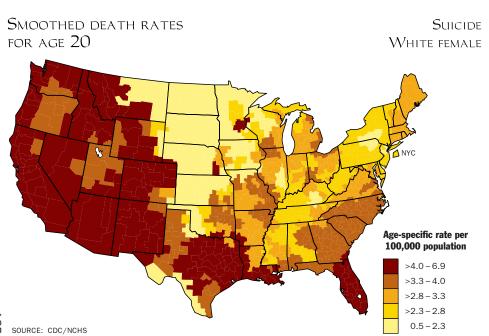
per 100,000 population

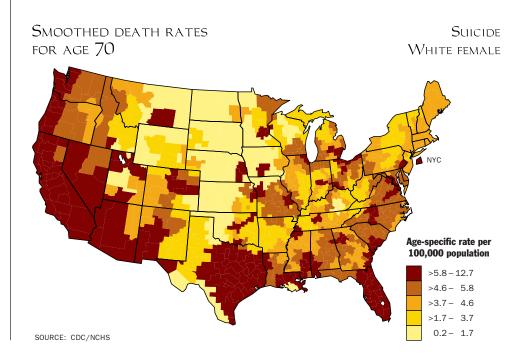


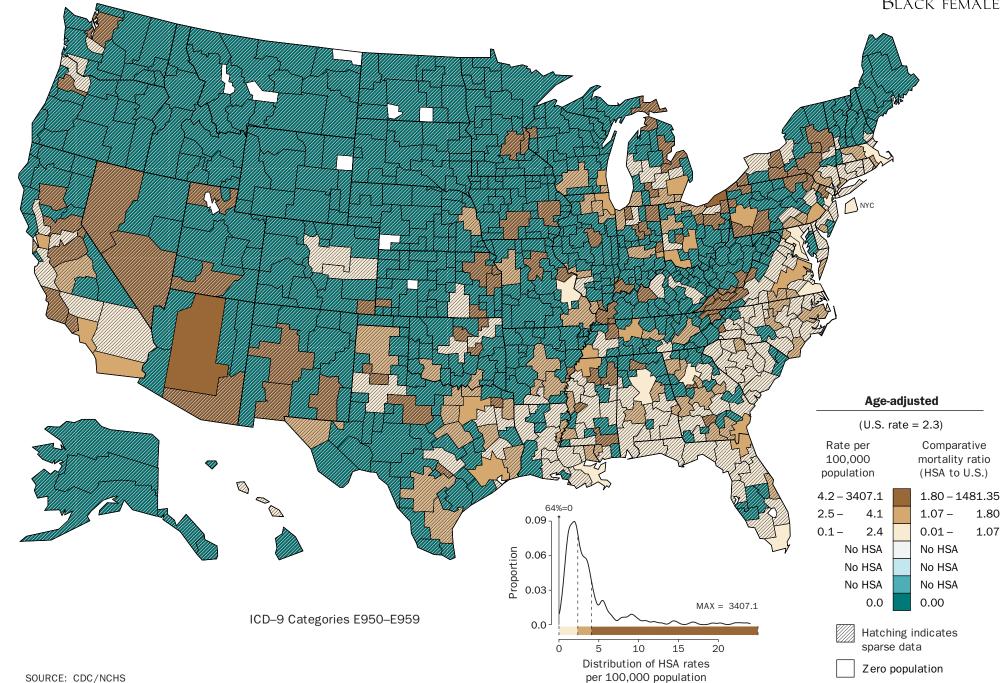


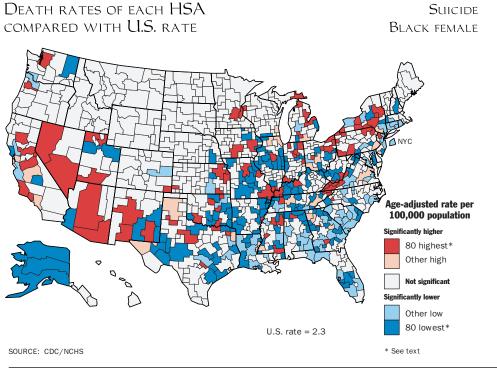




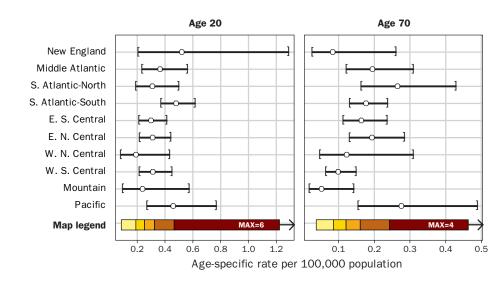


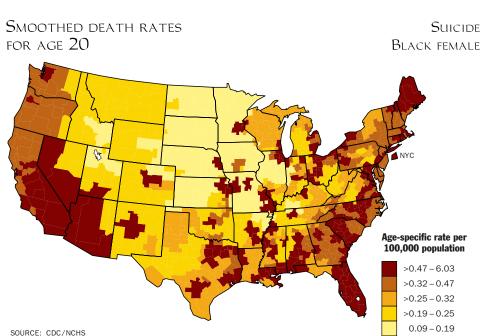


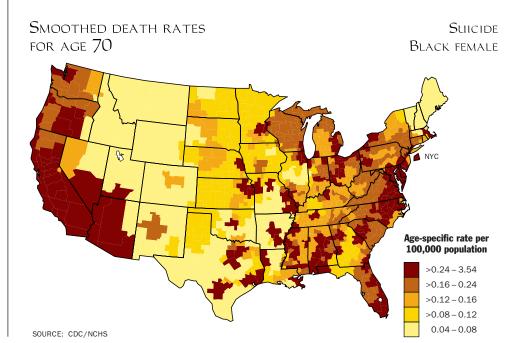


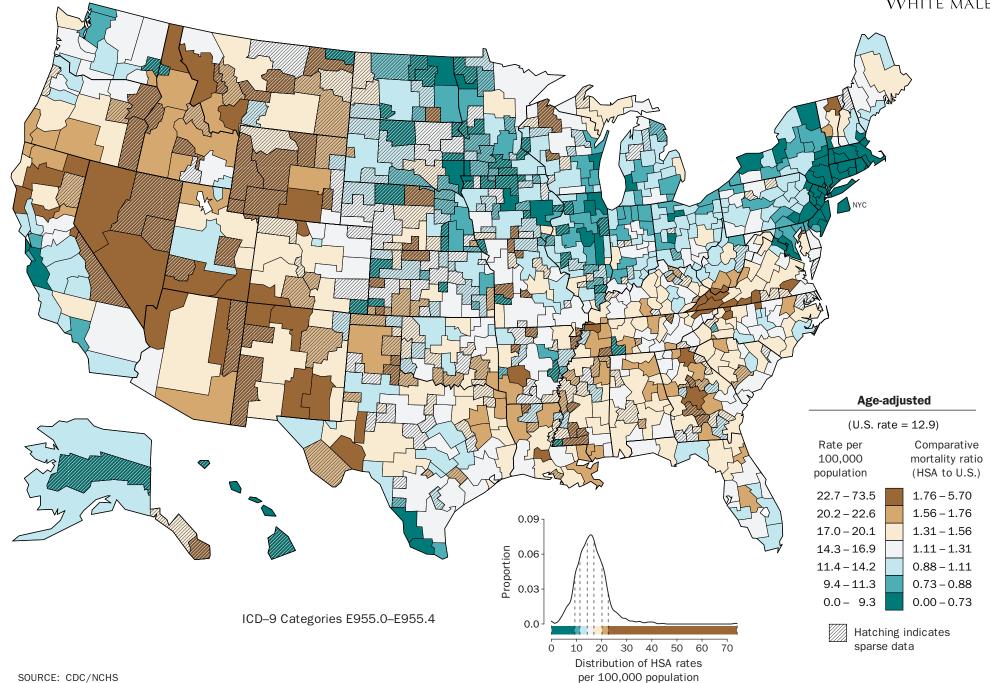


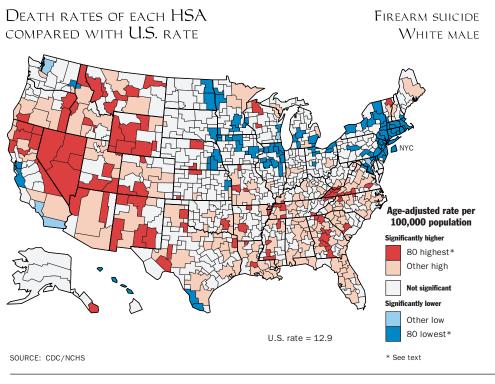
Suicide Black female





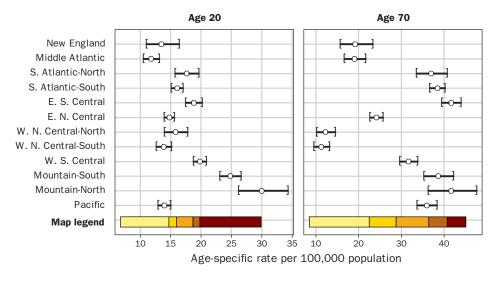


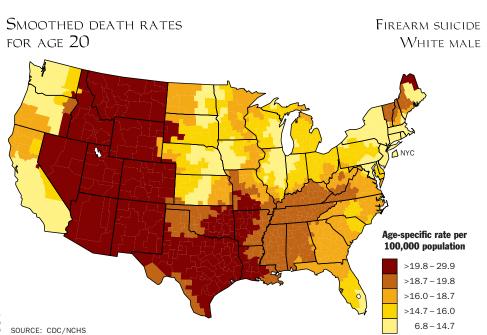


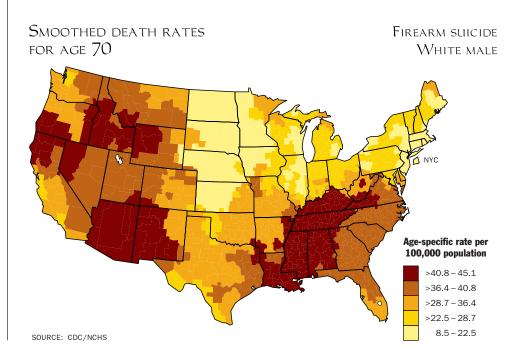


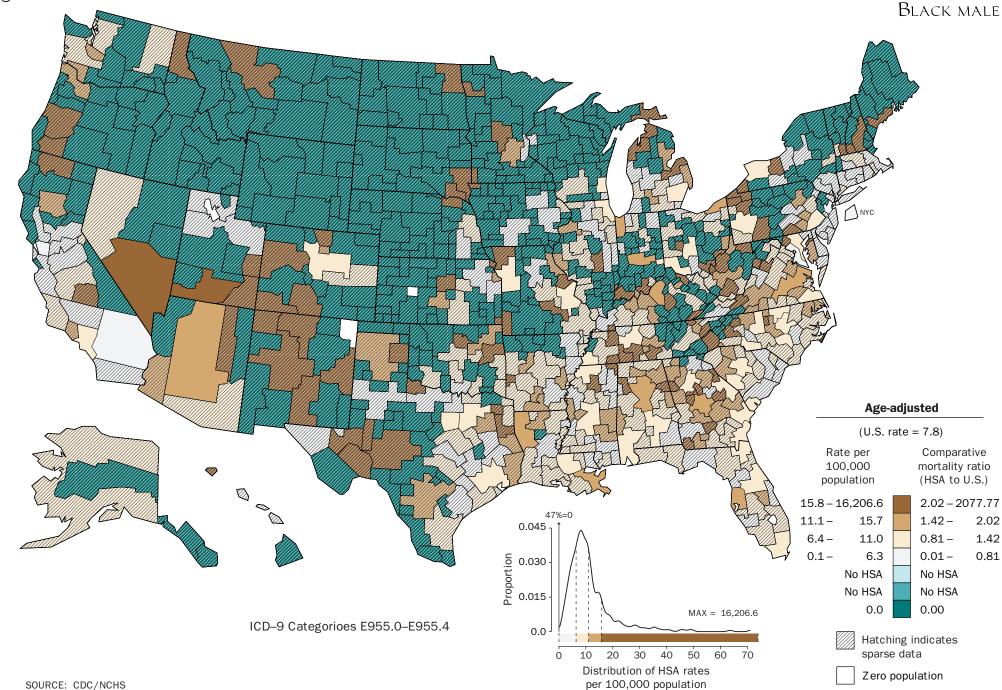


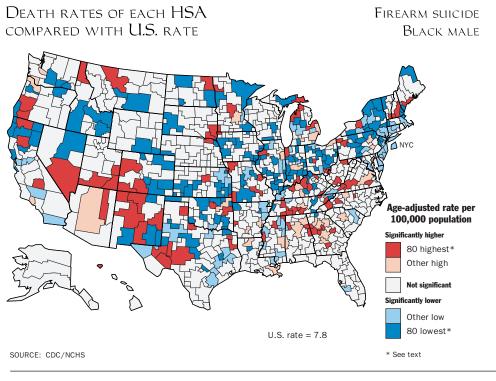
FIREARM SUICIDE WHITE MALE



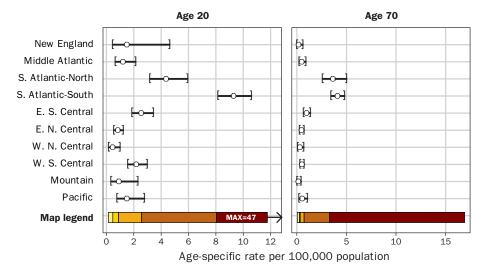


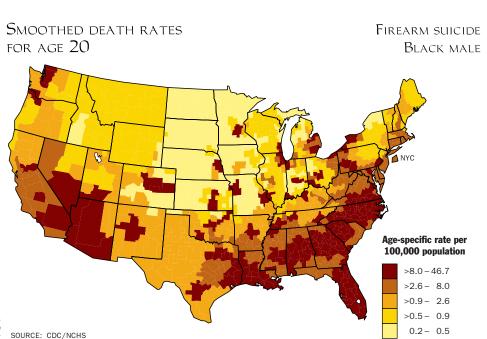


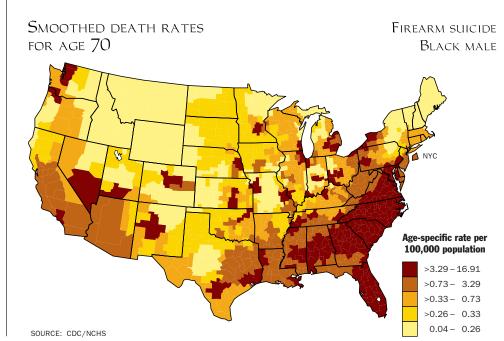




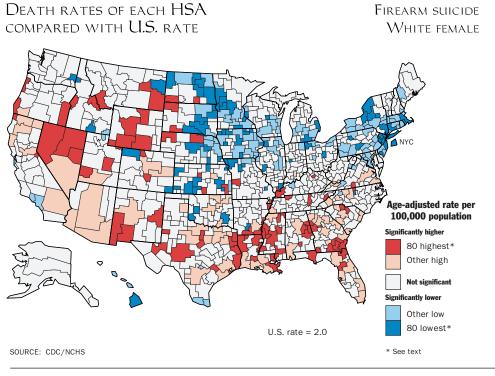
FIREARM SUICIDE BLACK MALE





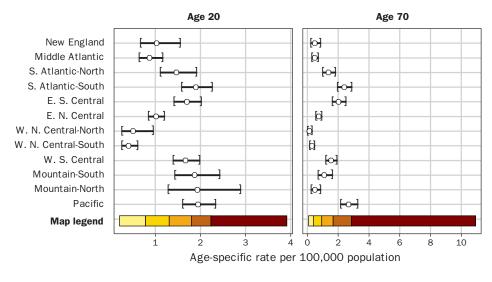


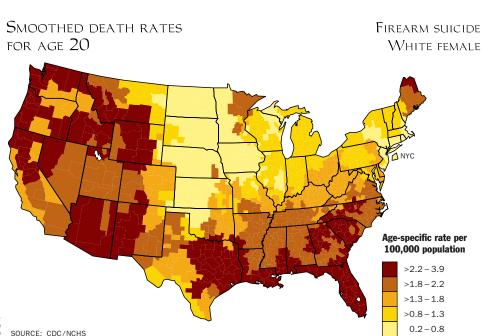
per 100,000 population

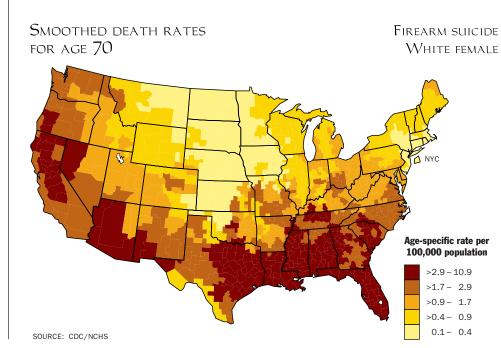


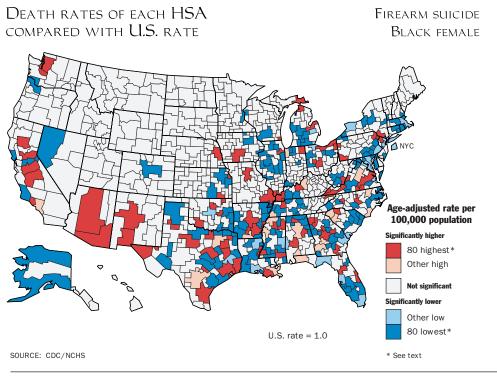


FIREARM SUICIDE WHITE FEMALE



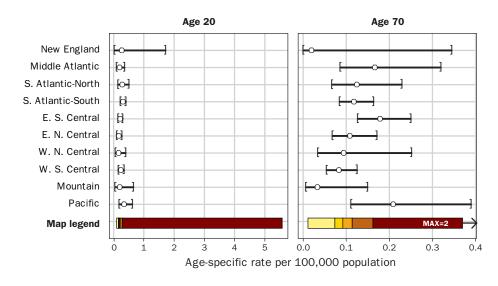




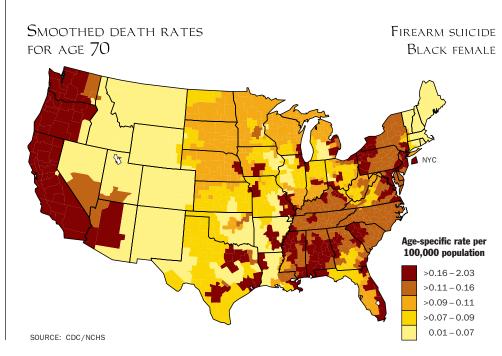




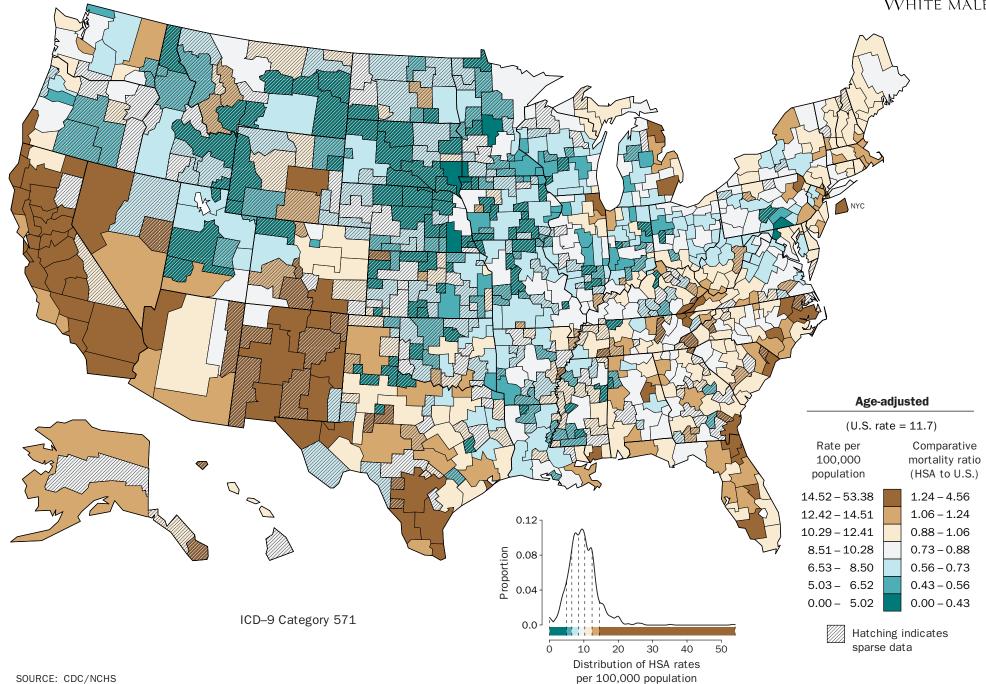
FIREARM SUICIDE BLACK FEMALE

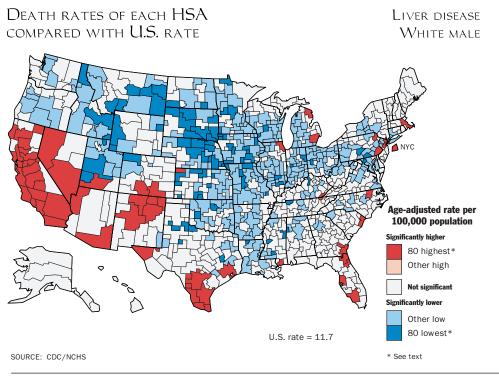


NOTE: Brackets indicate 95% confidence limits. SOURCE: CDC/NCHS



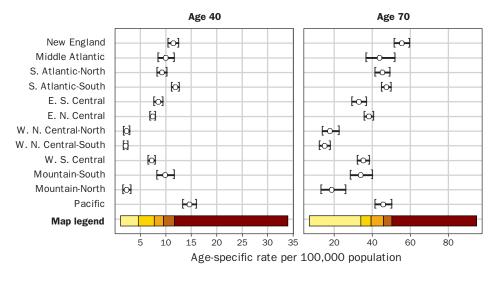


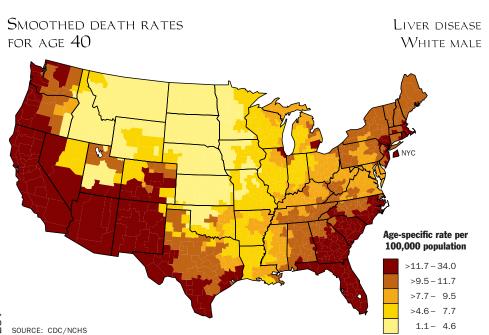


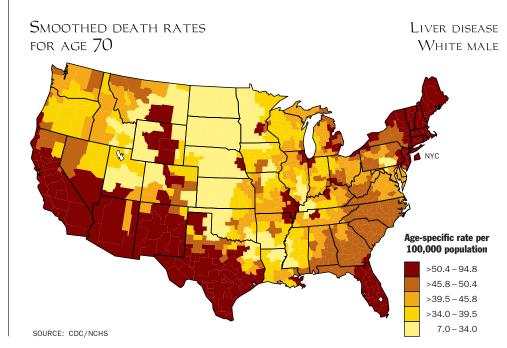


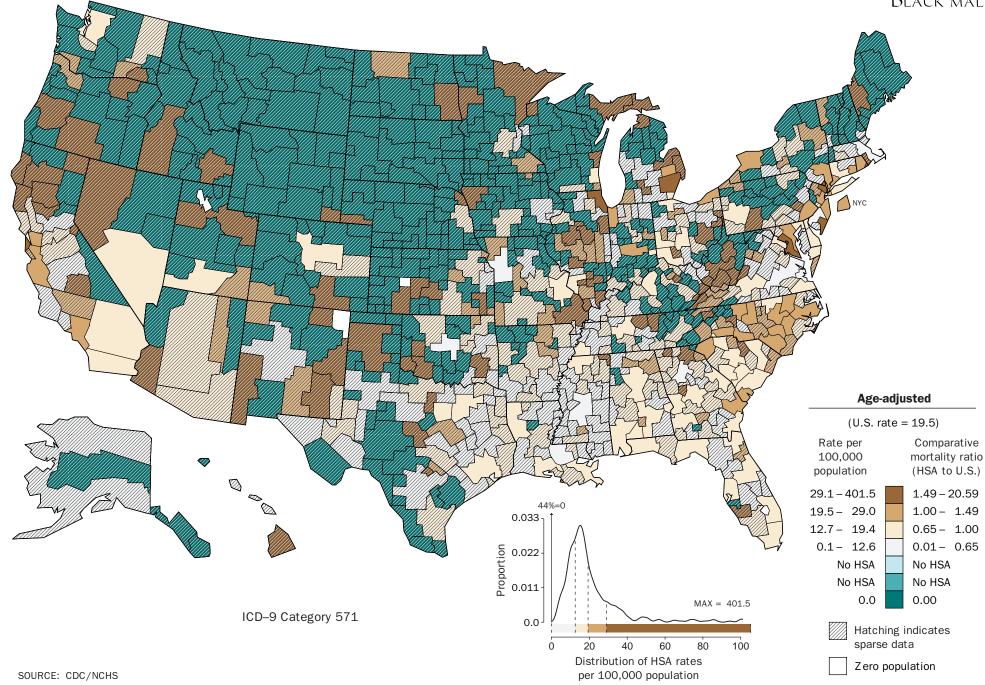
Predicted regional rates FOR SMOOTHED RATE MAPS

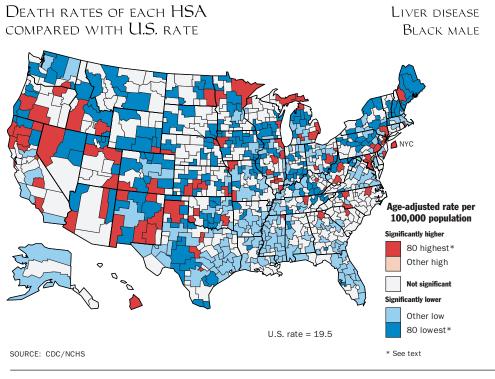
LIVER DISEASE White male



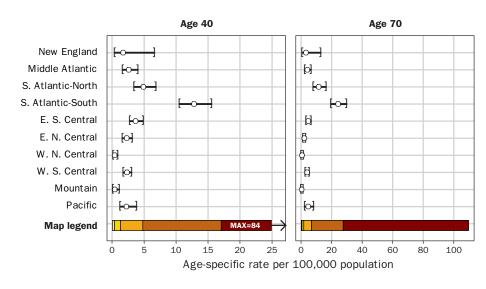


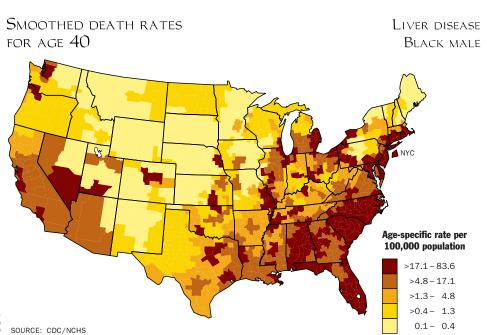


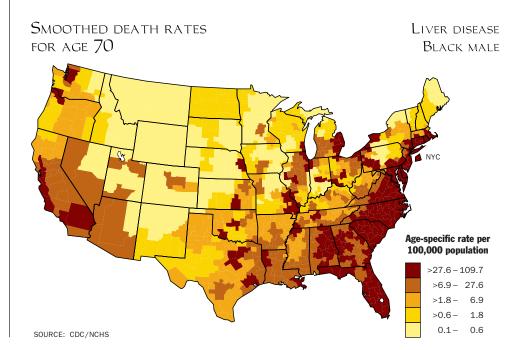


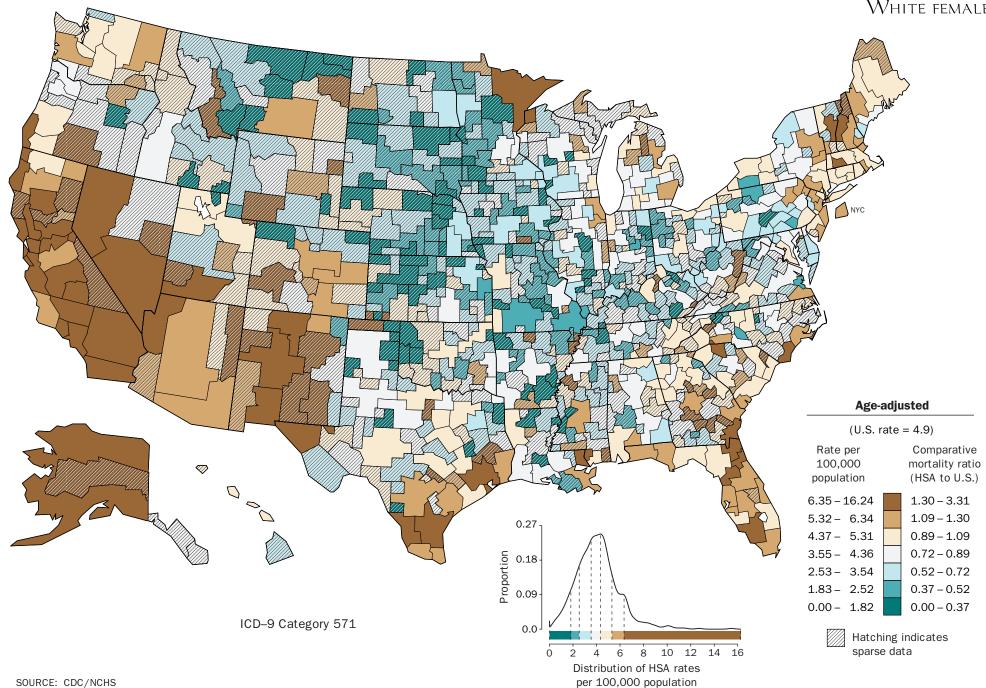


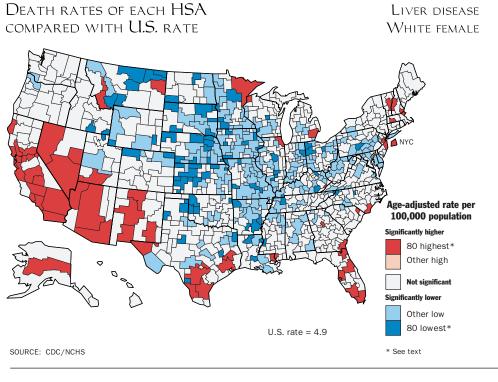
LIVER DISEASE BLACK MALE



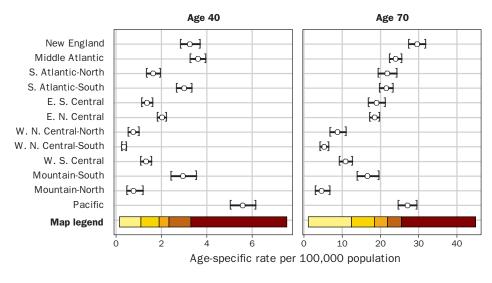


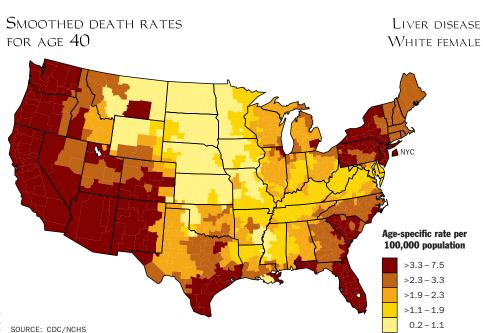


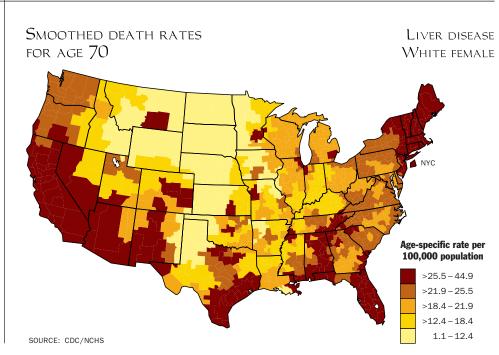




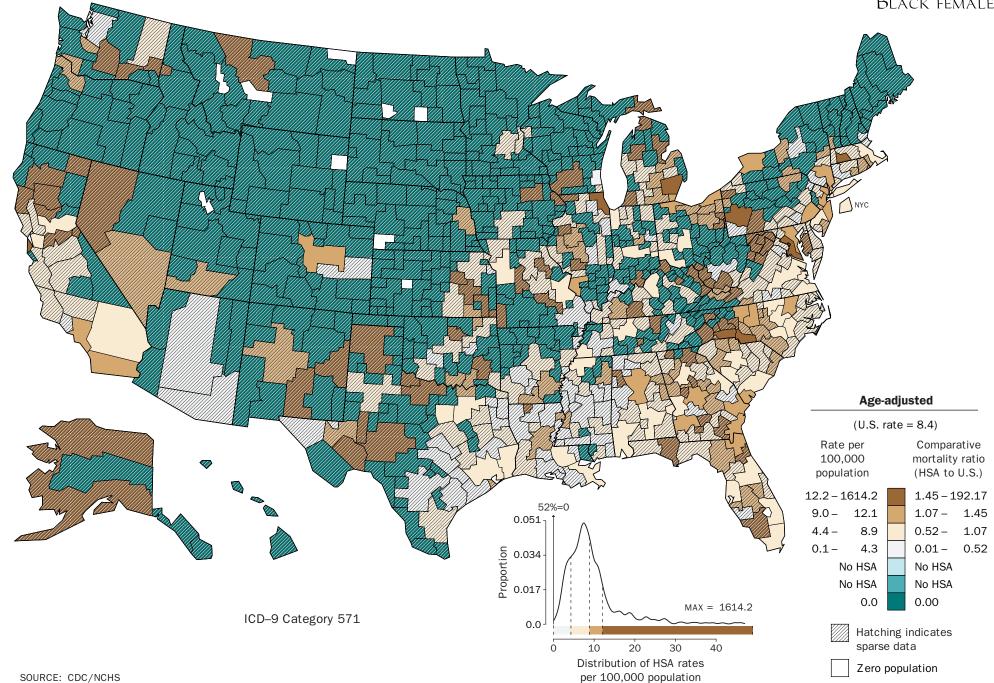
Liver disease White female

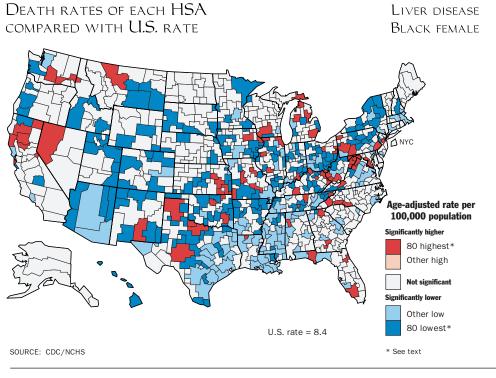




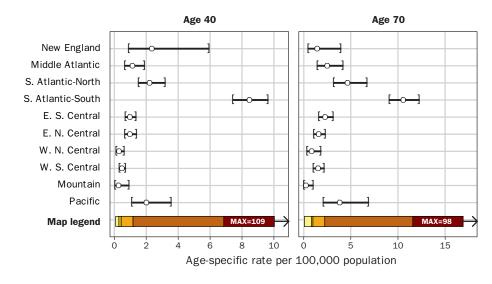


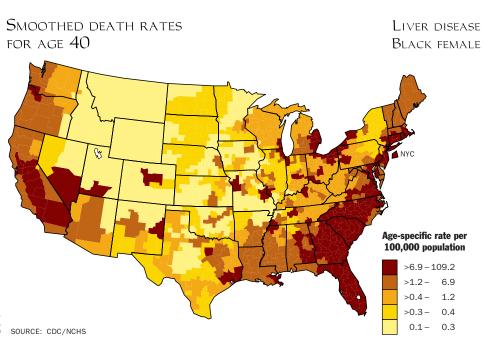


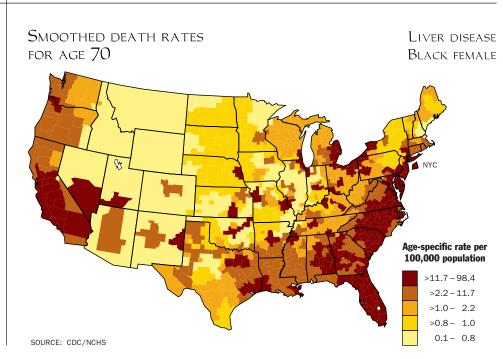




LIVER DISEASE BLACK FEMALE

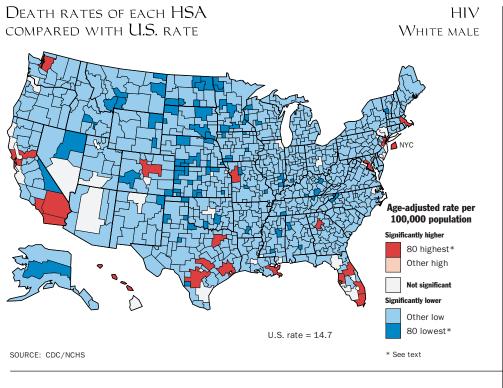




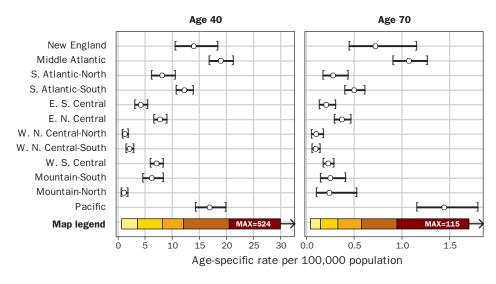


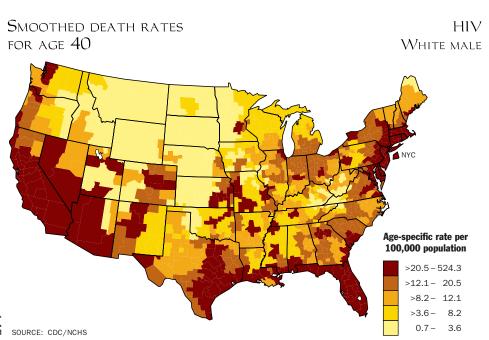
SOURCE: CDC/NCHS

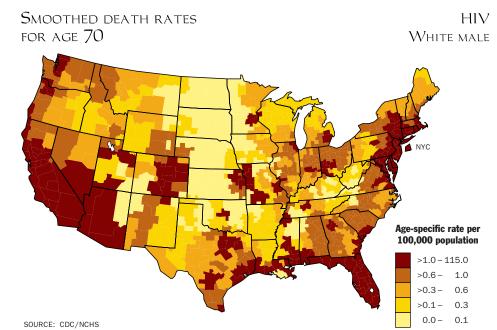
per 100,000 population

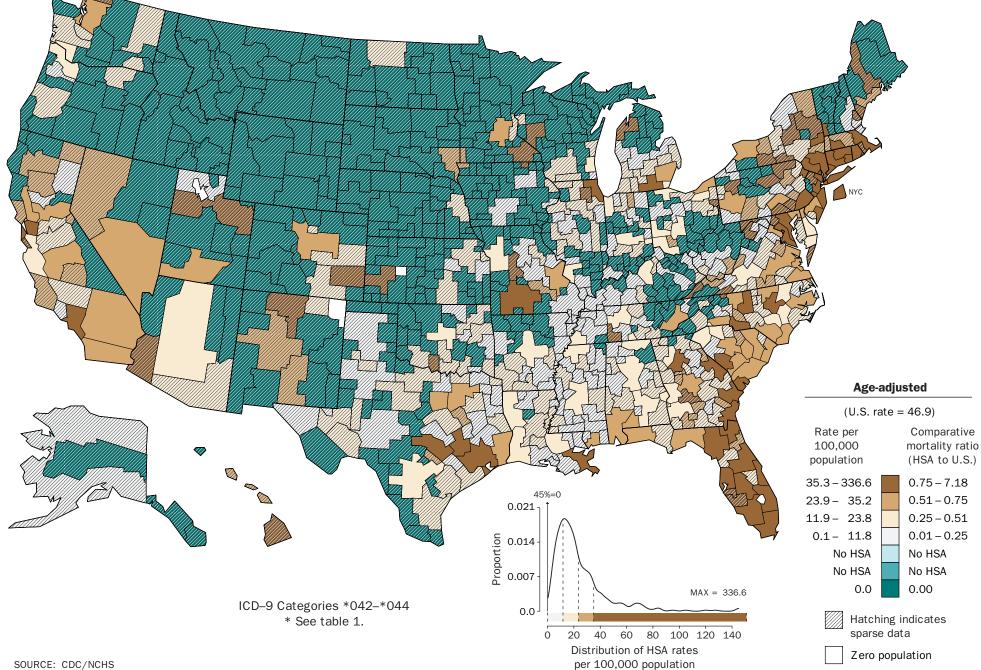


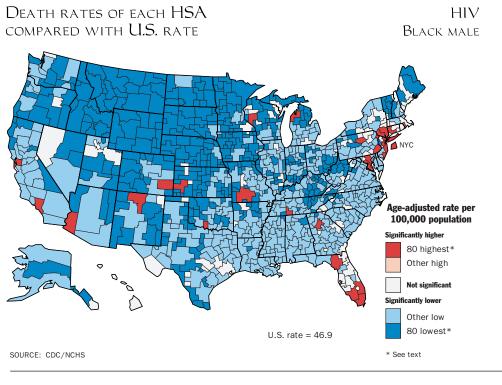






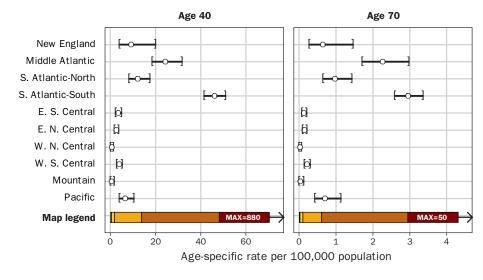


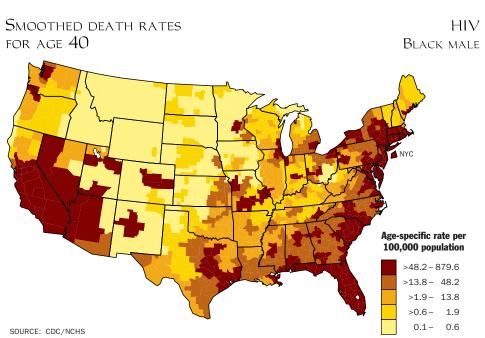


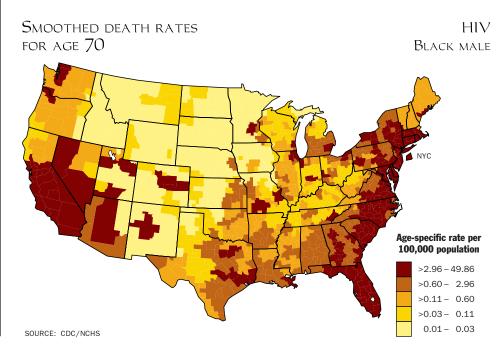


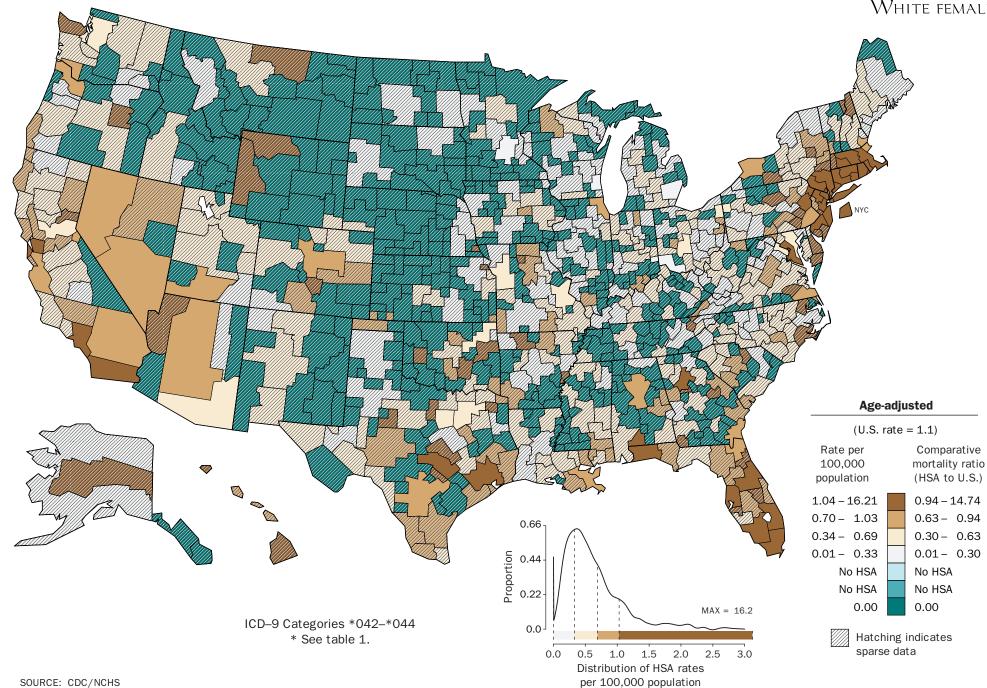


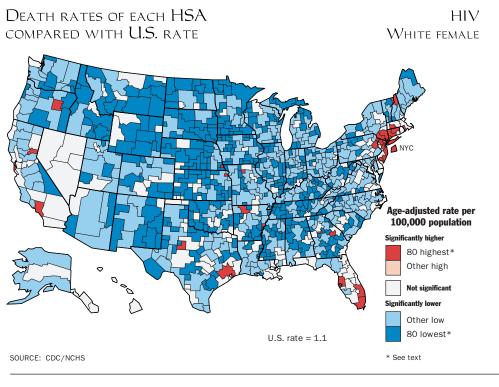






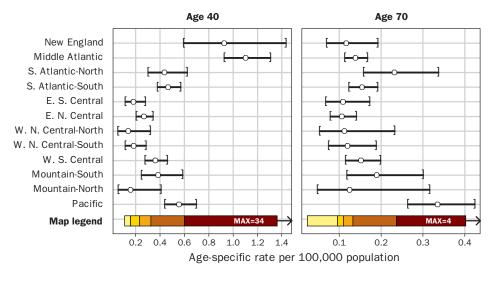


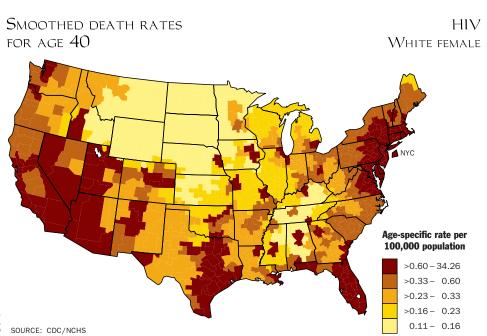


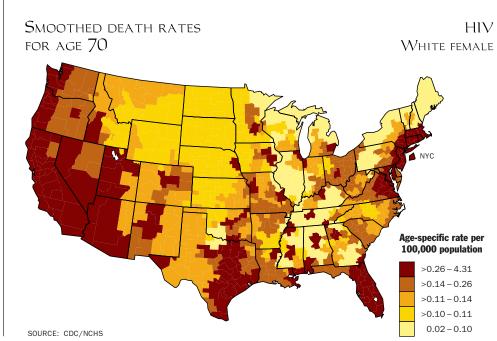


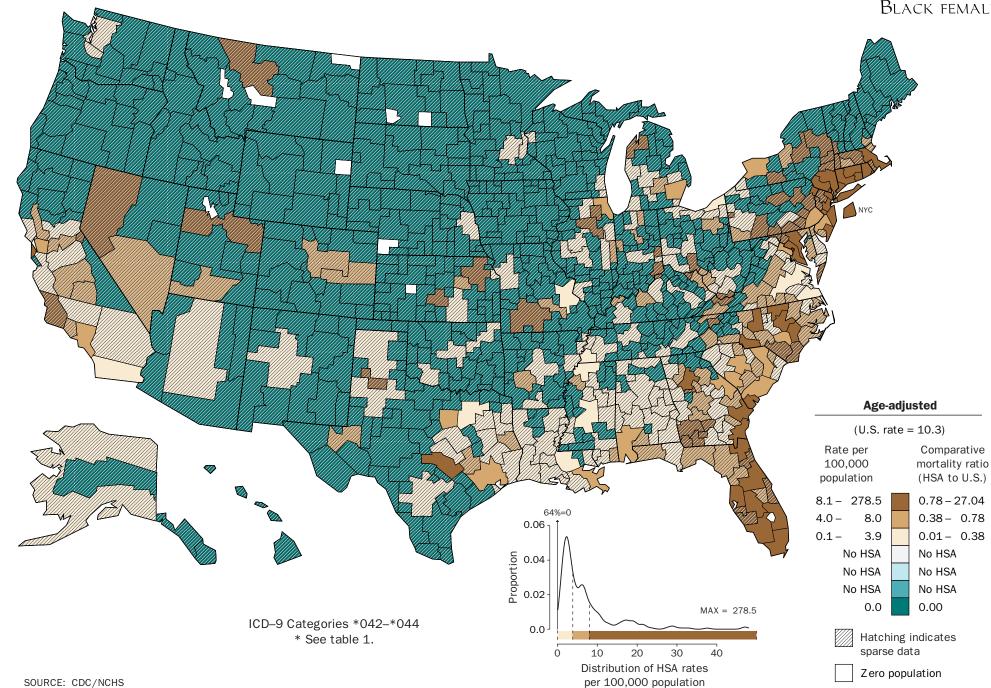


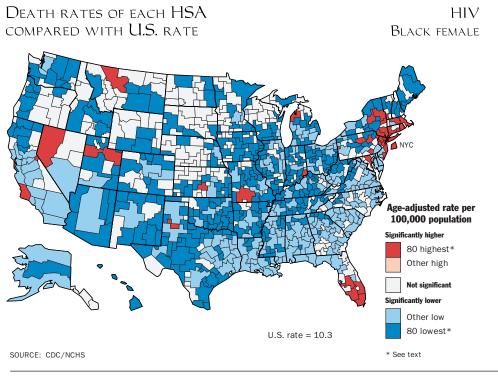




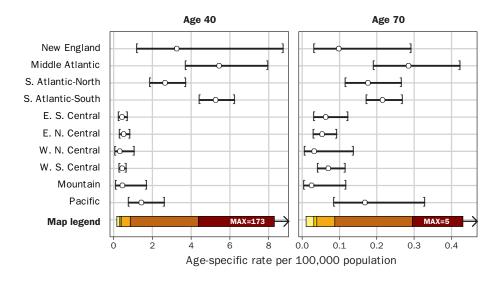


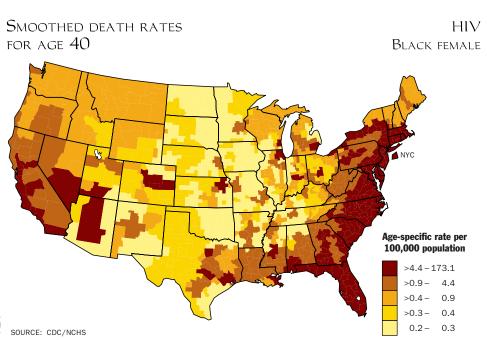


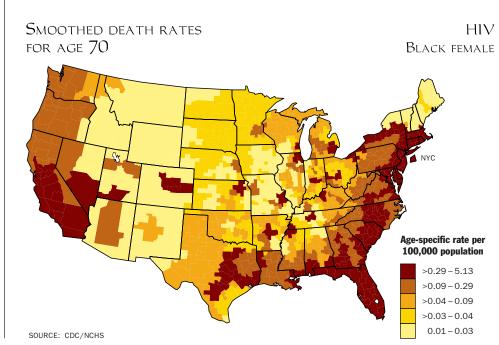




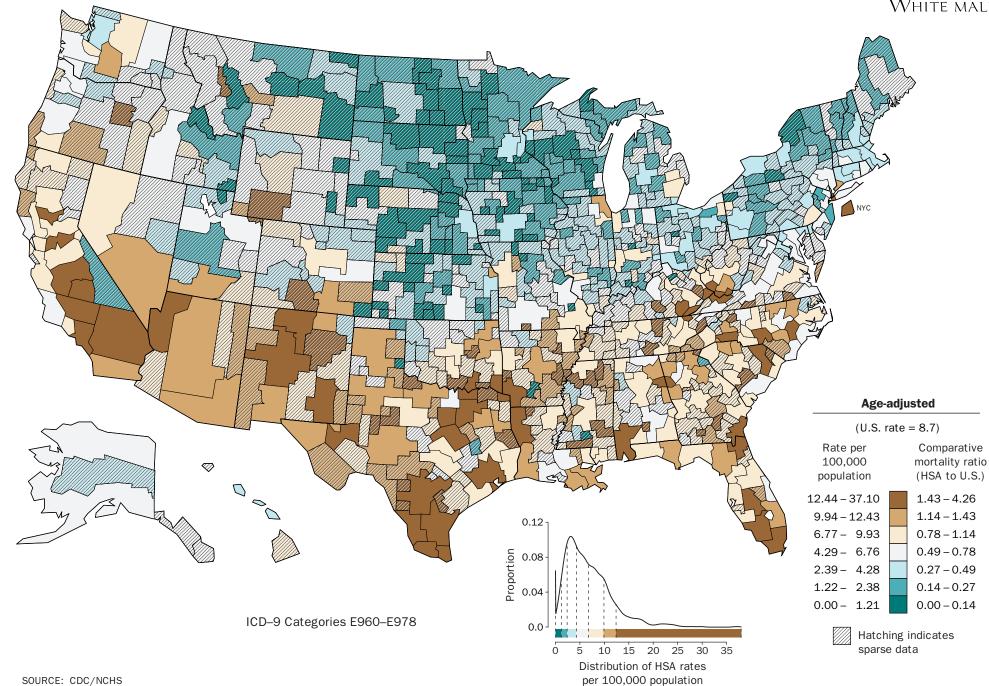


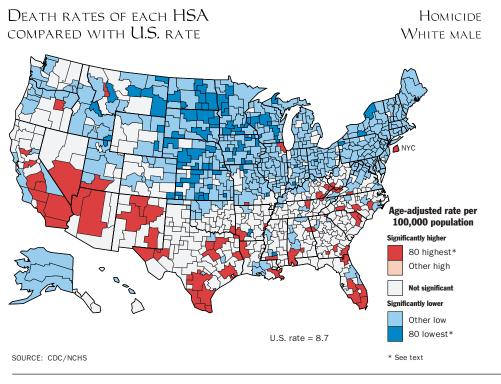






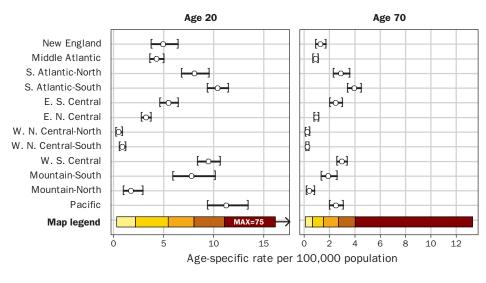
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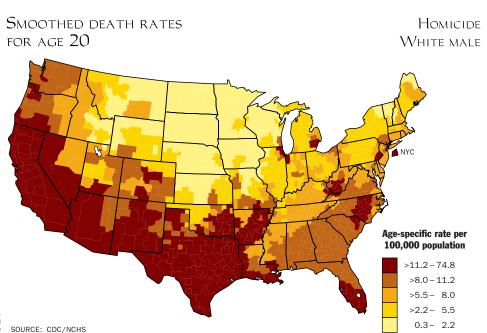


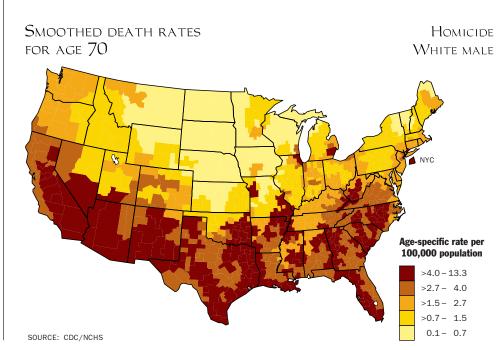


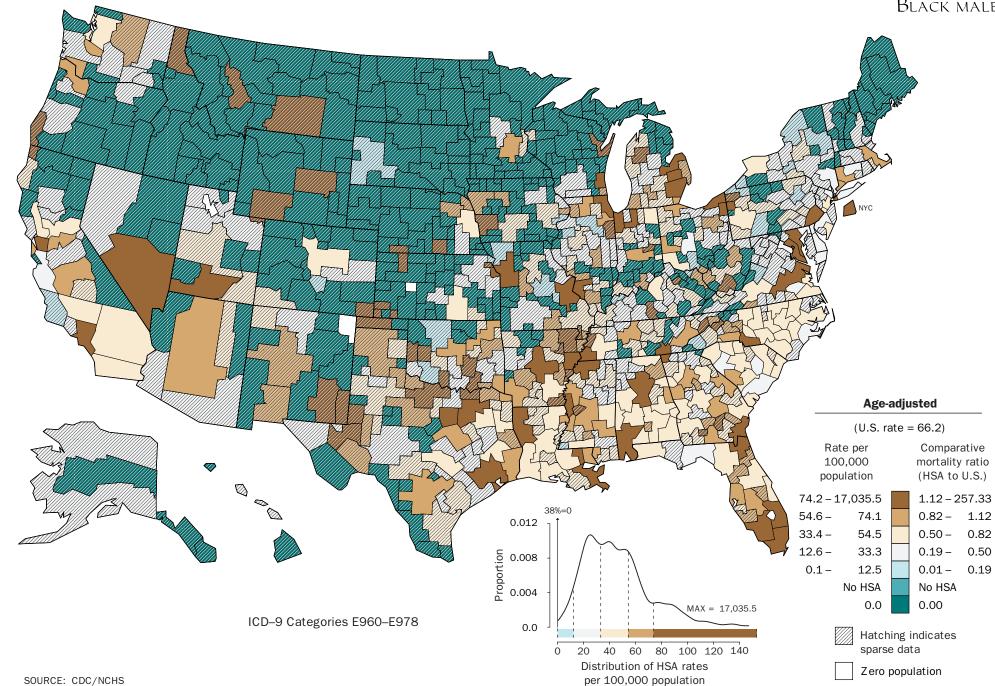


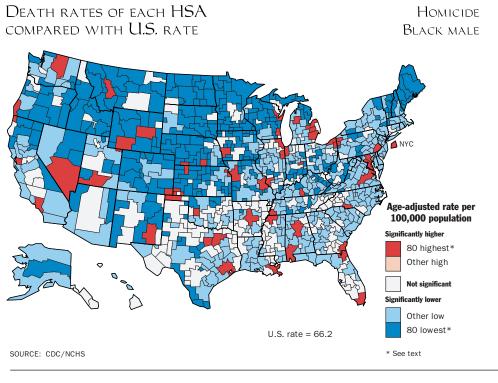




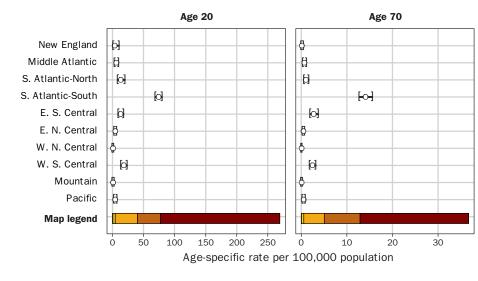


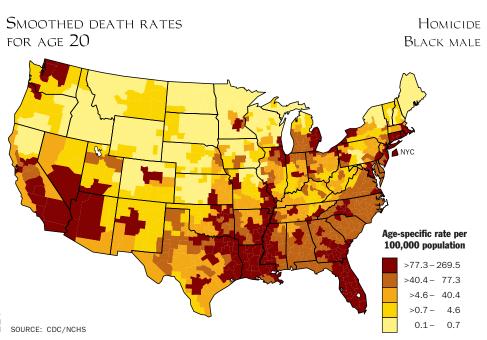


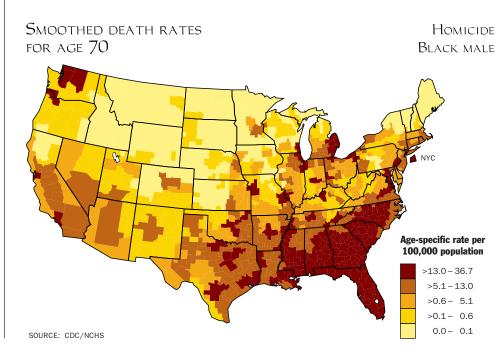




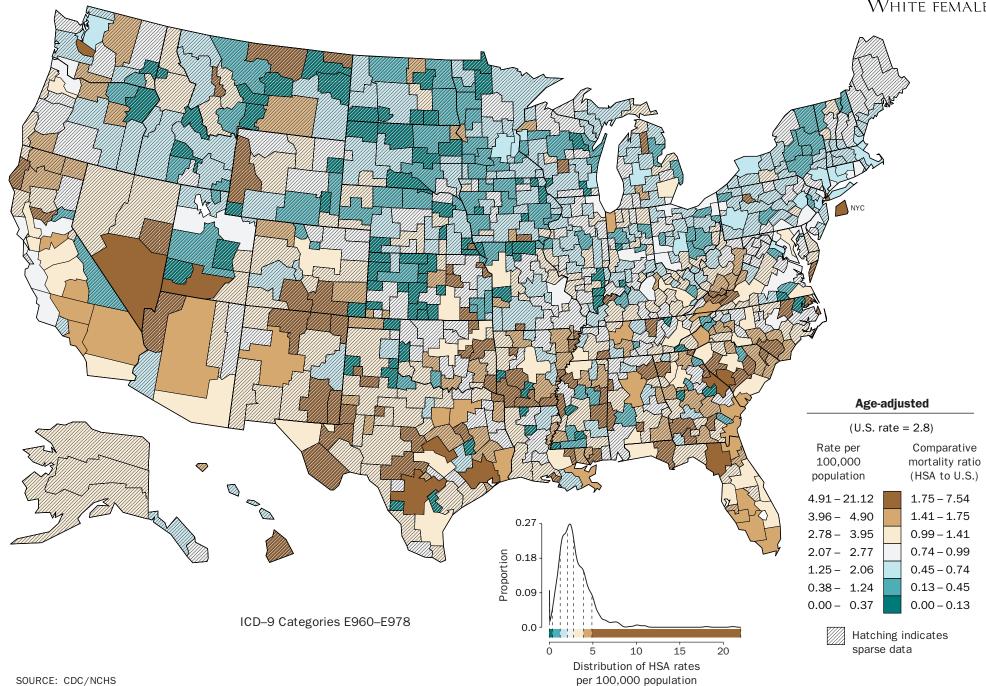


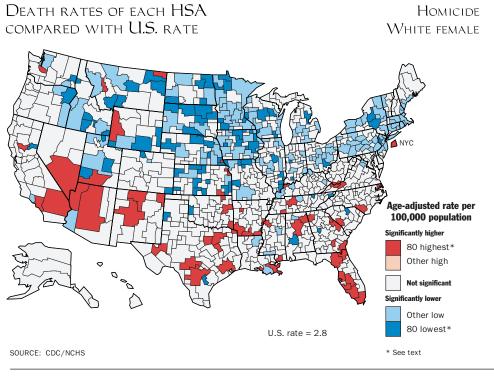




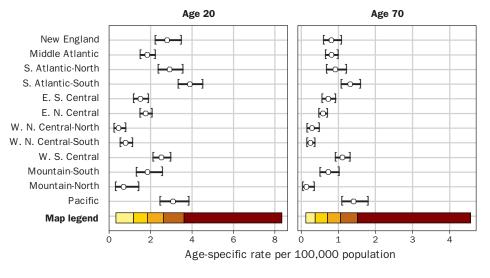


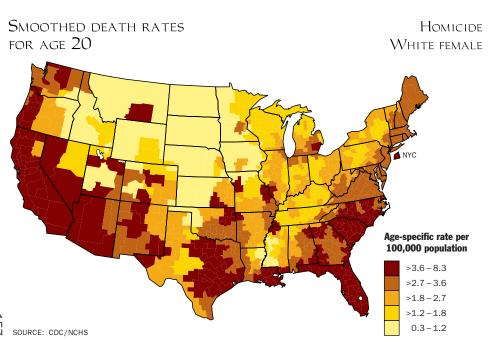


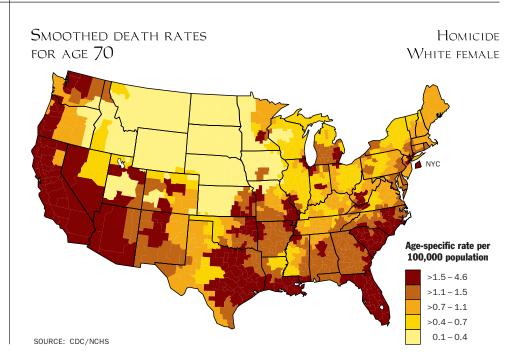


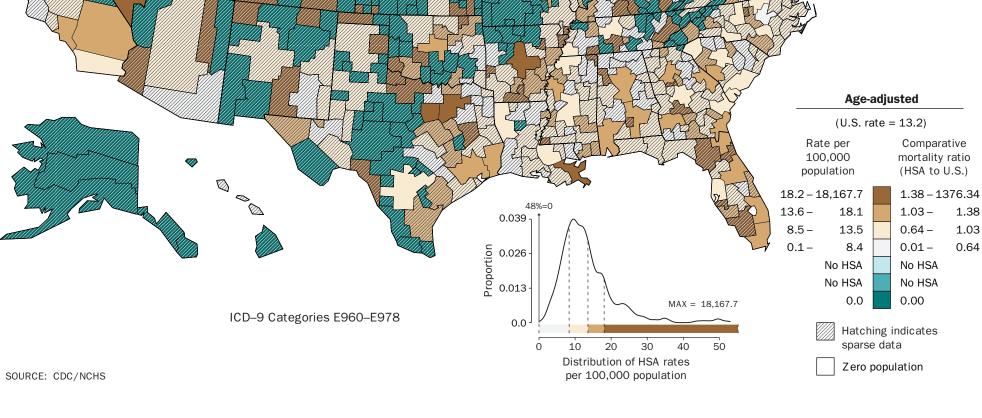


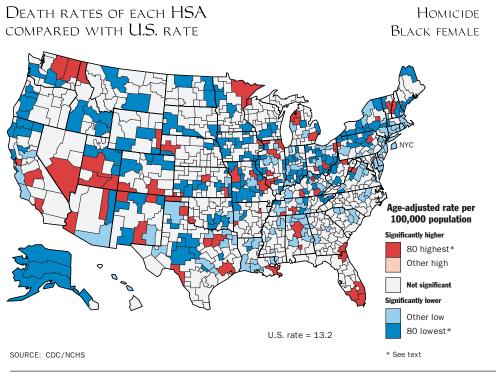
Homicide White female



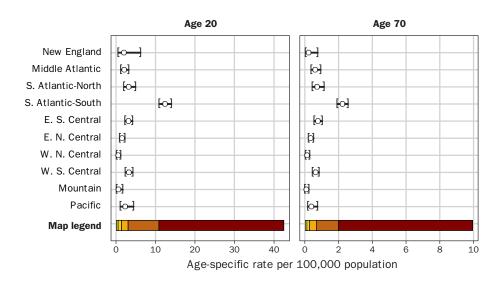


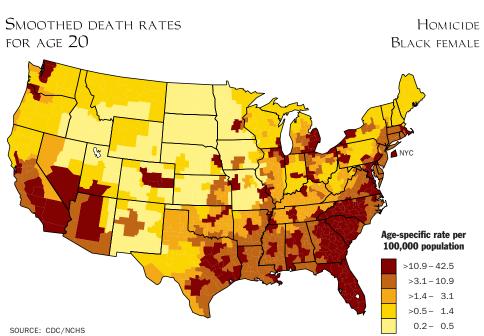


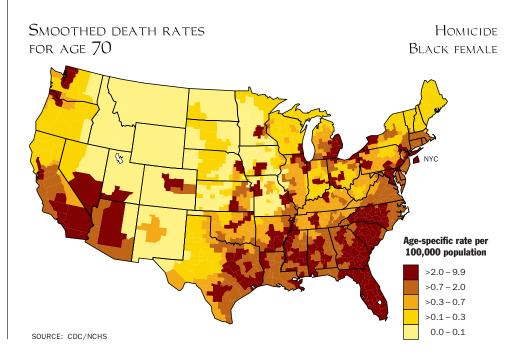






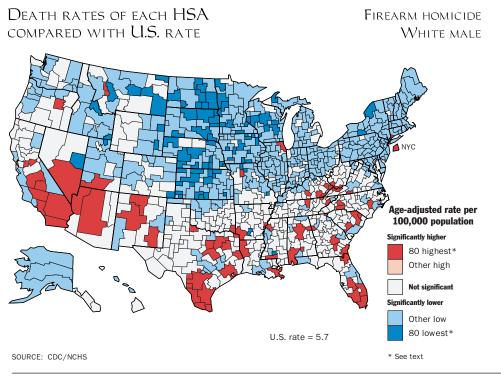






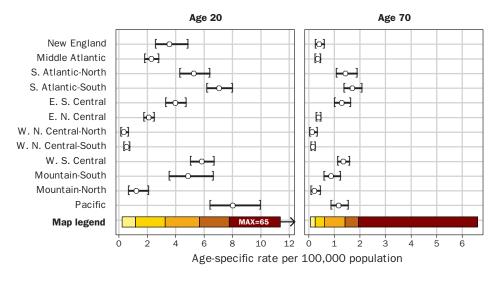
Distribution of HSA rates per 100,000 population

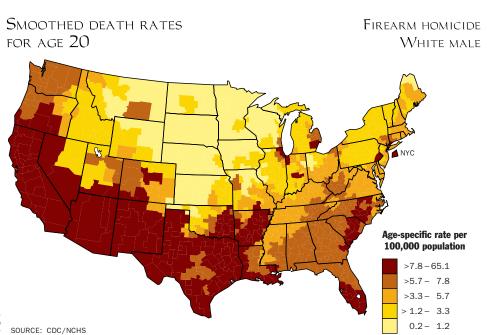
SOURCE: CDC/NCHS

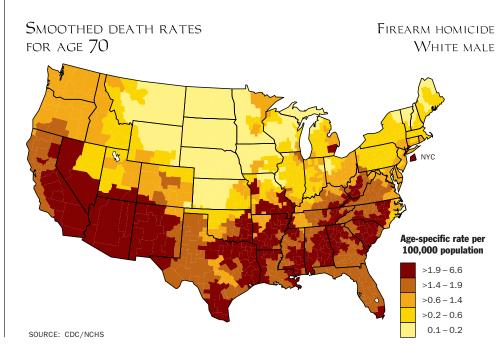




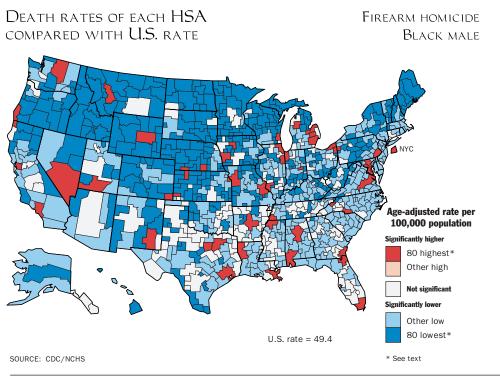
FIREARM HOMICIDE WHITE MALE





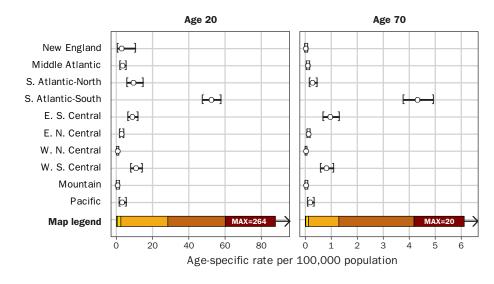


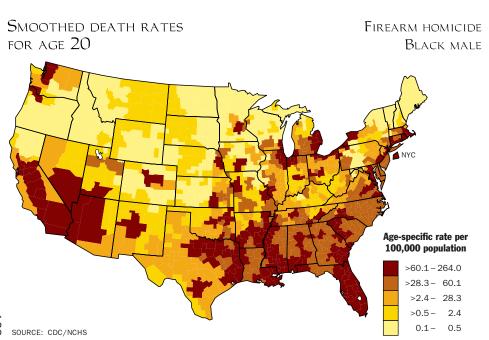
sparse data 20 60 80 Distribution of HSA rates Zero population per 100,000 population SOURCE: CDC/NCHS

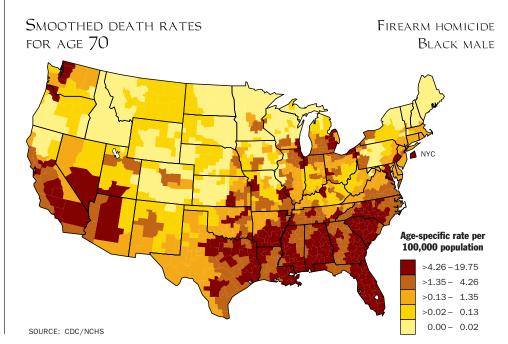


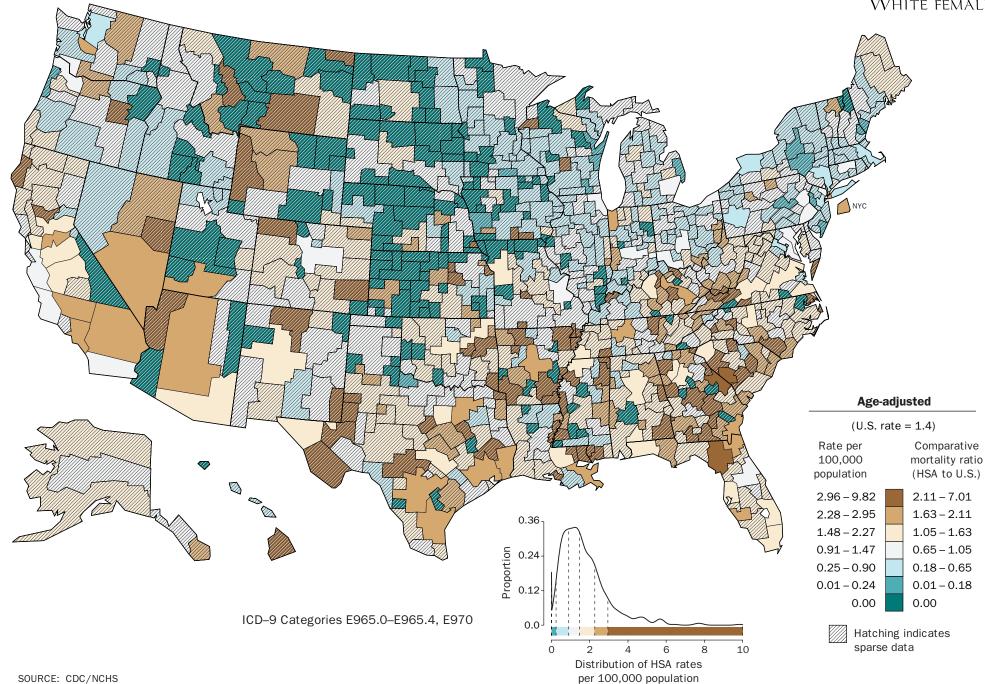


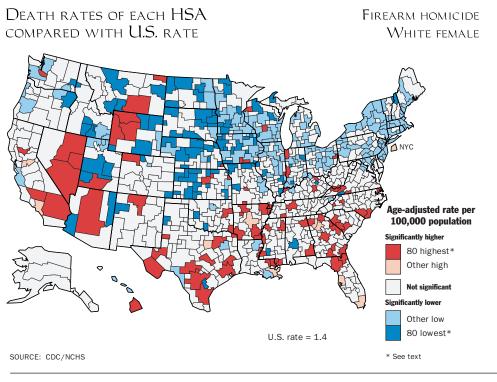
FIREARM HOMICIDE
BLACK MALE





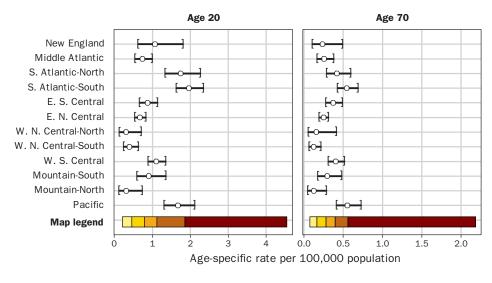


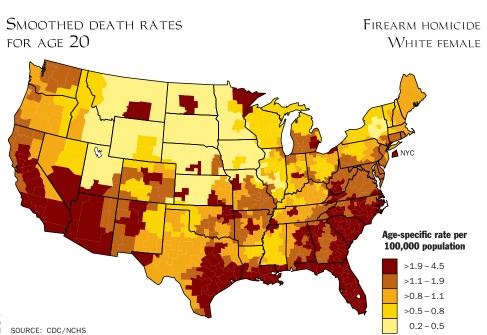


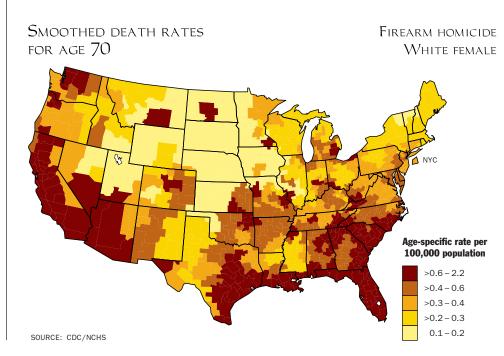


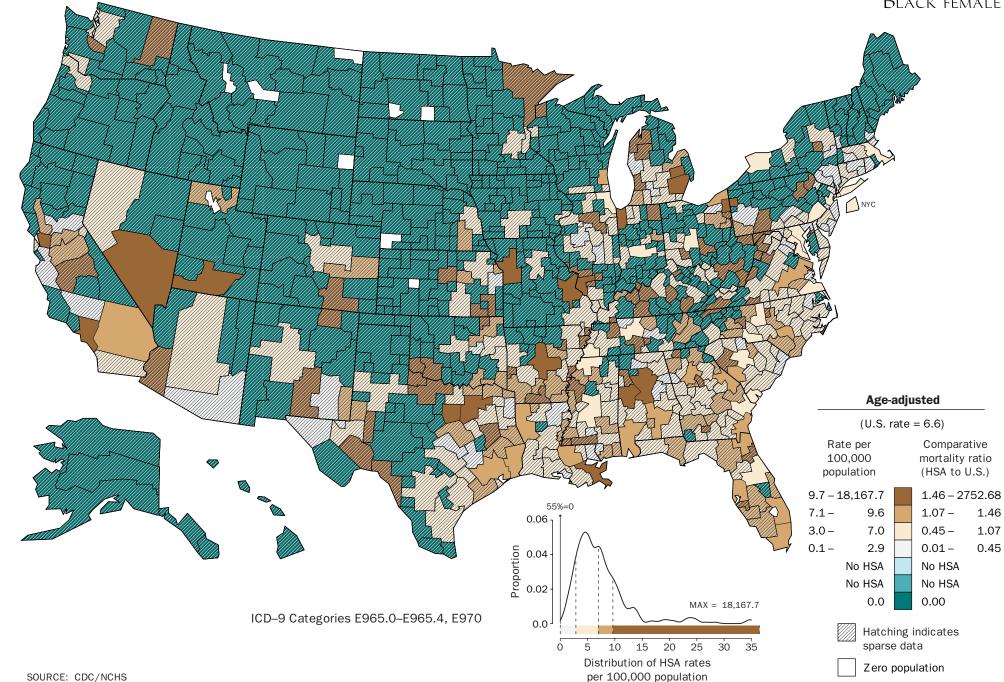


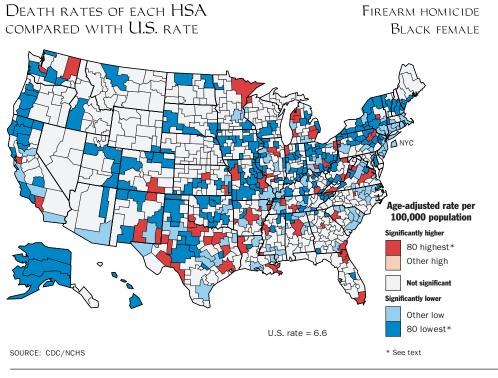
Firearm homicide White female





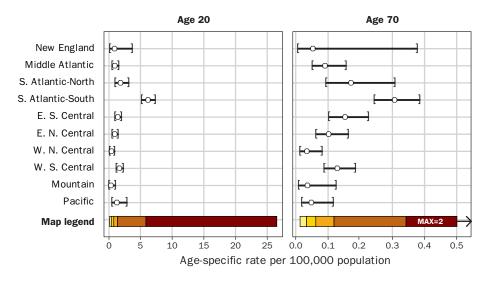


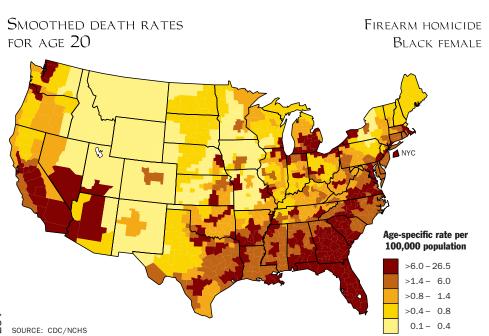


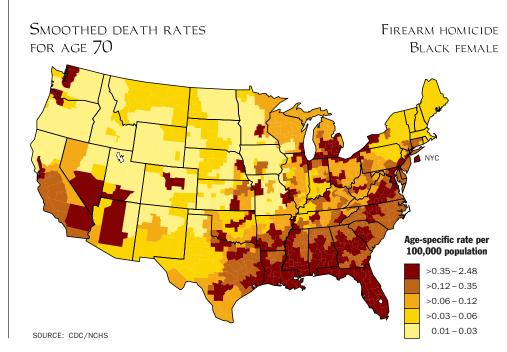


PREDICTED REGIONAL RATES FOR SMOOTHED RATE MAPS

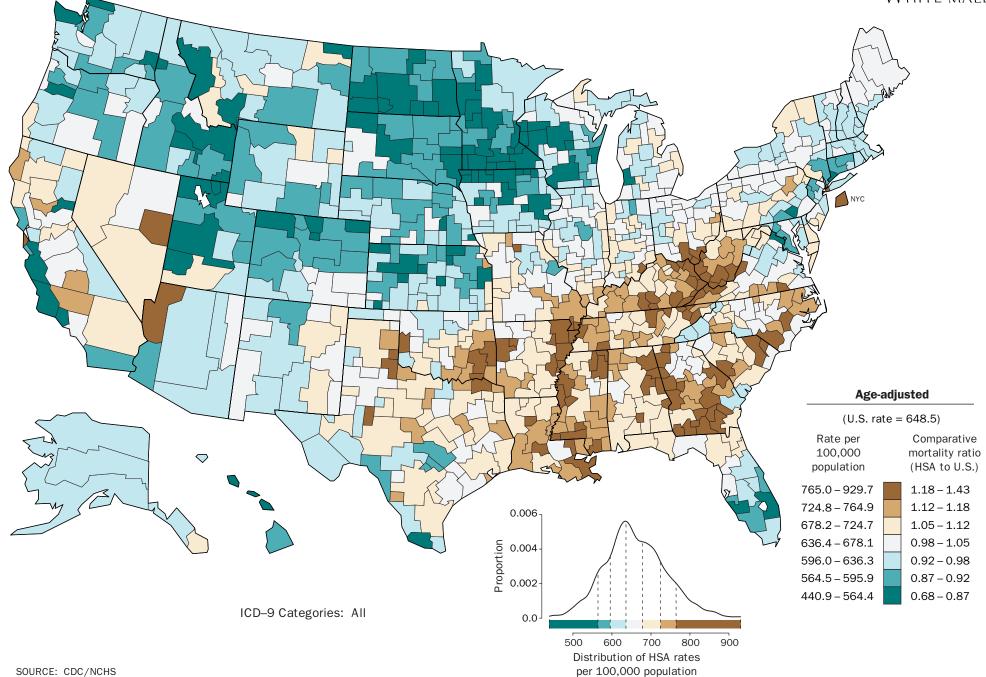
FIREARM HOMICIDE BLACK FEMALE

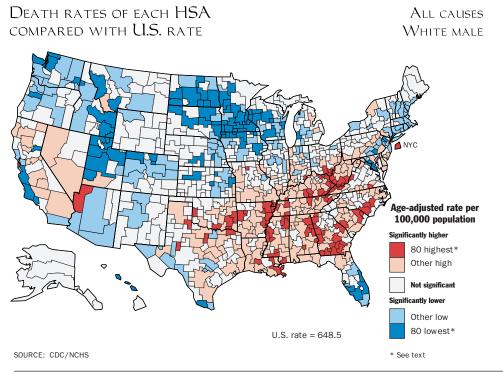






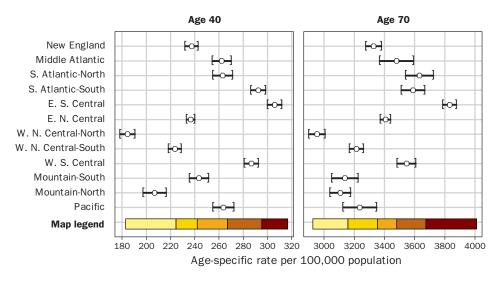


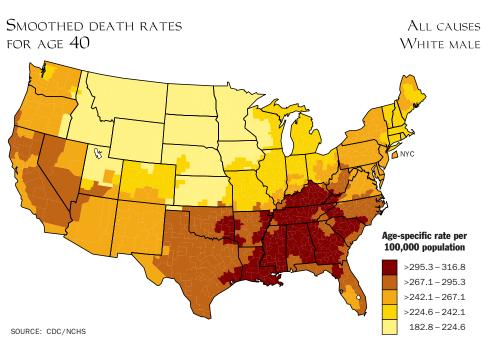


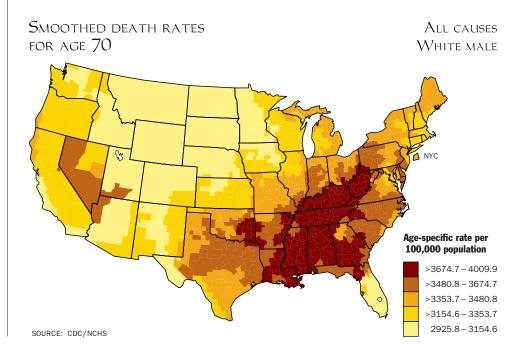


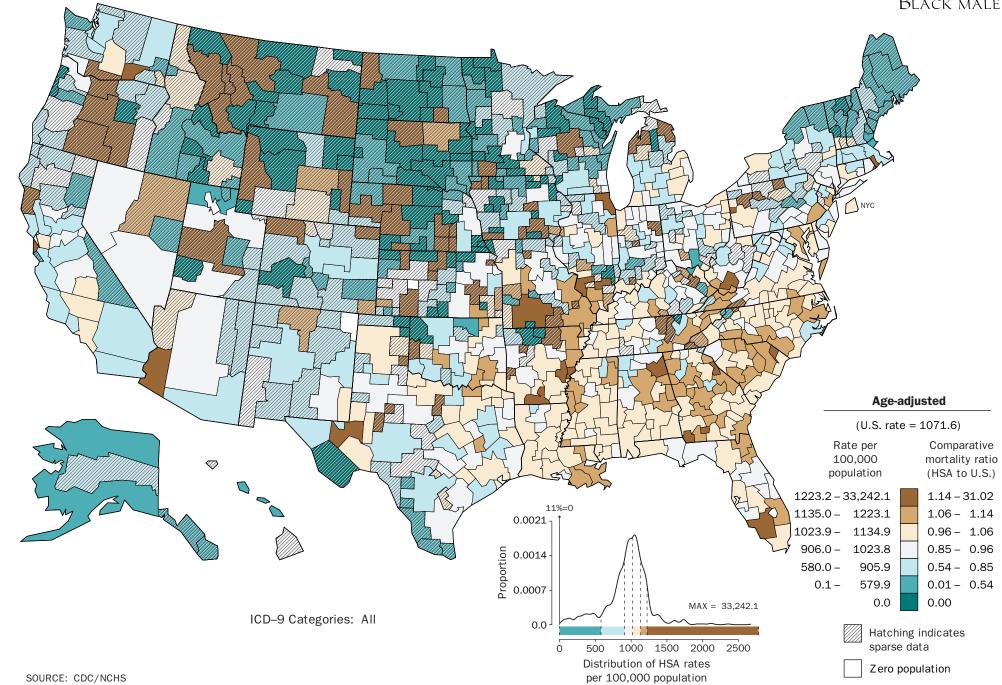


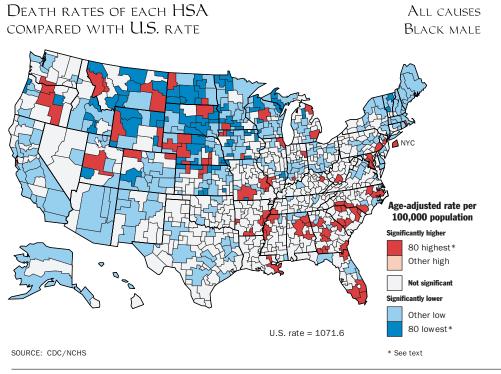
All causes White male

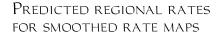




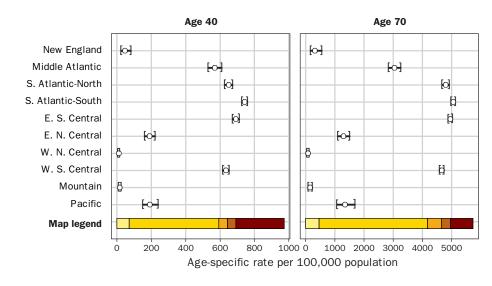


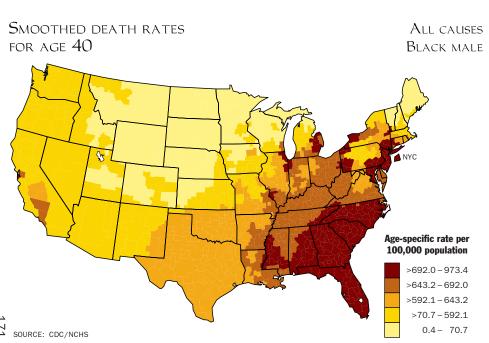


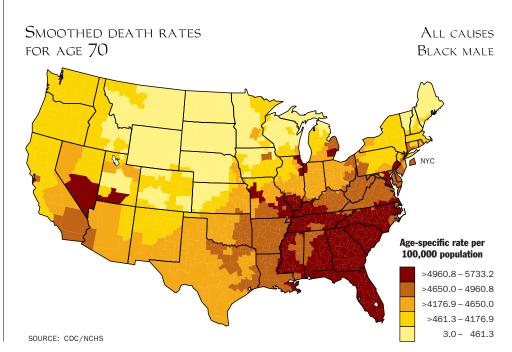




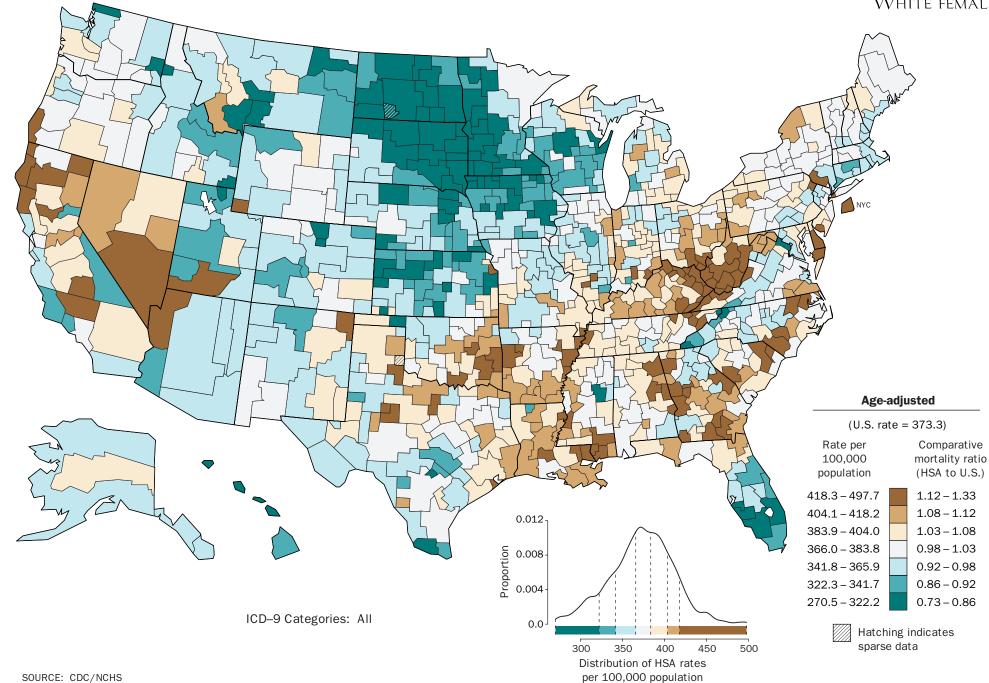
ALL CAUSES
BLACK MALE

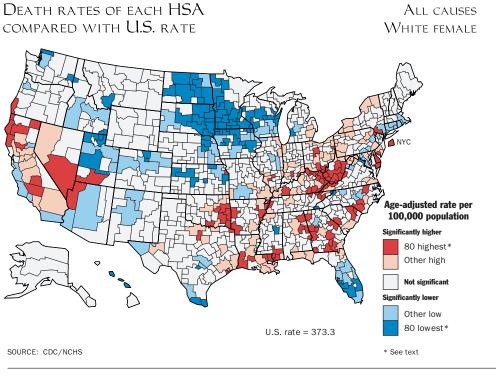




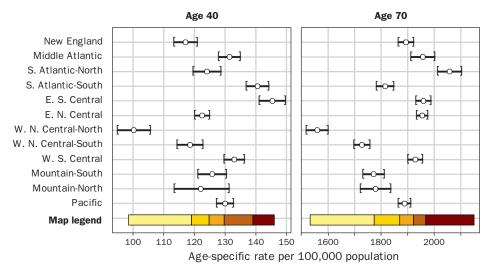


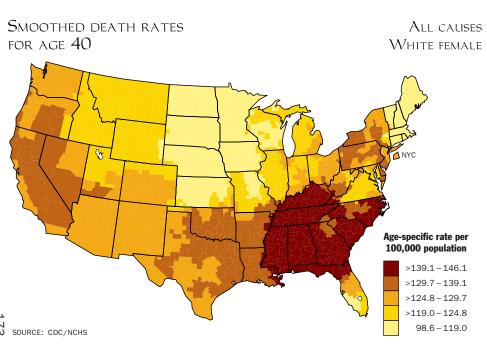
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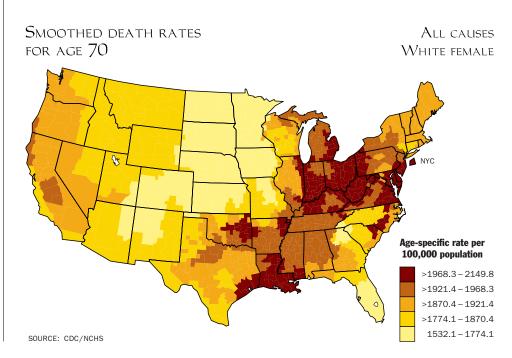


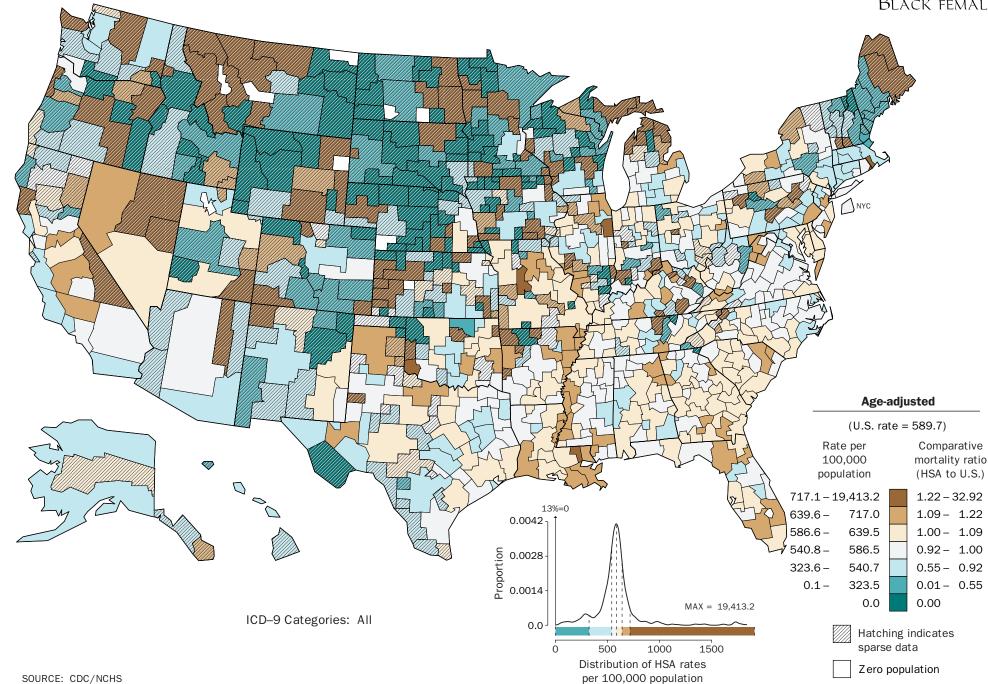


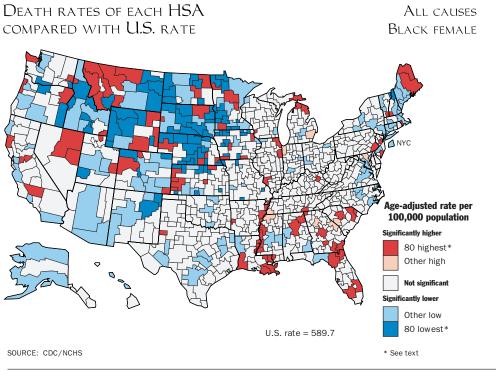
All causes White female



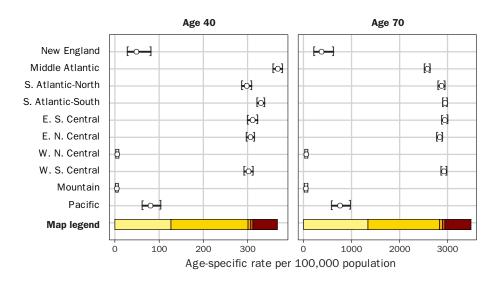


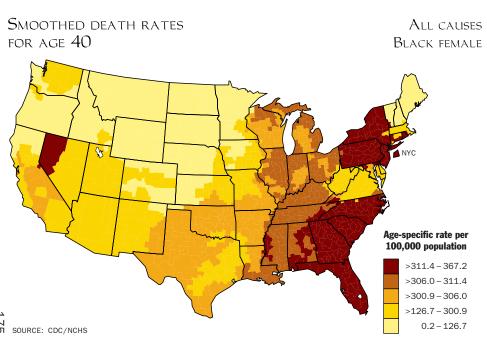


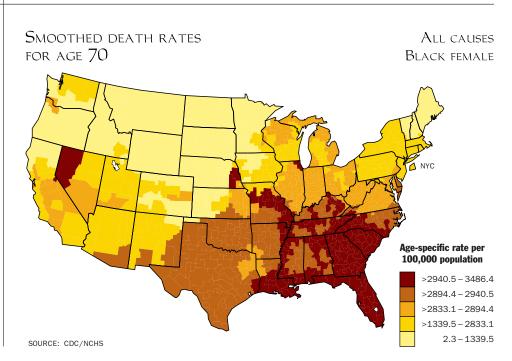




ALL CAUSES
BLACK FEMALE







- 1. Shattuck L. Art. XVI-Report to the committee of the city council. Am J Med Sci 12:177–8. 1846.
- 2. Snow J. On the mode of communication of cholera. 2d ed. New York: The Commonwealth Fund. 1855.
- Mason TJ, McKay FW, Hoover R, et al. Atlas of cancer mortality for U.S. counties: 1950–1969. Washington: USGPO (DHEW pub no (NIH) 75–780). 1975.
- 4. Blot WJ, Harrington M, Toledo A, et al. Lung cancer after employment in shipyards during World War II. N Engl J Med 299:620–4. 1978.
- 5. Winn DM, Blot WJ, Shy CM, et al. Snuff dipping and oral cancer among women in the southern United States. N Engl J Med 304:745–9. 1981.
- Mason TJ, McKay FW, Hoover R, et al. Atlas of cancer mortality among U.S. nonwhites: 1950– 1969. Washington: USGPO (DHEW pub no (NIH) 76–1204). 1976.
- 7. Mason TJ; Fraumeni JF, Jr.; Hoover R; et al. An atlas of mortality from selected diseases. Washington: USGPO (DHHS pub no (NIH) 81–2397). 1981.
- 8. Pickle LW, Mason TJ, Howard N, et al. Atlas of U.S. cancer mortality among whites: 1950–1980. Washington: USGPO (DHHS pub no (NIH) 87–2900). 1987.
- 9. Pickle LW, Mason TJ, Howard N, et al. Atlas of U.S. cancer mortality among nonwhites: 1950–1980. Washington: USGPO (DHHS pub no (NIH) 90–1582). 1990.
- Walter SD, Birnie SE. Mapping mortality and morbidity patterns: An international comparison. Int J Epidemiol 20:678–89. 1991.
- 11. Pickle LW, Herrmann DJ, eds. Cognitive aspects of statistical mapping. Working paper series report no 18. Hyattsville, Maryland: National Center for Health Statistics. 1995.
- 12. Devine OJ, Annest JL, Kirk ML, et al. Injury mortality atlas of the United States, 1979–1987. Atlanta: Centers for Disease Control. 1991.

- 13. National Center for Health Statistics. Monthly vital statistics report; vol 43 nos 1–12. Hyattsville, Maryland: National Center for Health Statistics. Public Health Service. 1995.
- 14. National Center for Health Statistics. Vital statistics of the United States, 1990, vol II, mortality, part A. Washington: Public Health Service. 1994.
- 15. U.S. Bureau of the Census. April 1, 1990, agerace-modified census counts.
- 16. World Health Organization. Manual of the International Statistical Classification of Diseases, Injuries, and Causes of Death, based on the recommendations of the Ninth Revision Conference, 1975. Geneva: World Health Organization. 1977.
- 17. Freedman MA. Health status indicators for the year 2000. Healthy people 2000: Statistical notes 1(1). Hyattsville, Maryland: DHHS pub no (PHS) 92–1237). 1991.
- 18. Gittelsohn A, Royston PN. Annotated bibliography of cause-of-death validation studies, 1958–80. National Center for Health Statistics. Vital Health Stat 2(89). 1982.
- Rosenberg HM. The nature and accuracy of cause-of-death data. Report of the Workshop on improving cause-of-death statistics. National Committee on Vital and Health Statistics. Hyattsville, Maryland: National Center for Health Statistics. 1989.
- 20. Harris KW, French DK. A methodological study of quality control procedures for mortality medical coding. Vital Health Stat 2(81). 1980. DHEW pub no (PHS) 79–1355. 1980.
- National Center for Health Statistics. Vital statistics, ICD–9 ACME decision tables for classifying underlying causes of death, 1990.
 NCHS instruction manual; part 2c. Hyattsville, Maryland: Public Health Service. Published annually.
- 22. Sirken MG, Rosenberg HM, Chevarley FM, Curtin LR. The quality of cause-of-death statistics. Am J Public Health 77:137–9. 1987.

- 23. Israel RA, Rosenberg HM, Curtin LR. Analytical potential for multiple cause-of-death data. Am J Epidemiol 124:161–79. 1986.
- 24. Gittelsohn A, Senning J. Studies on the reliability of vital and health records: I. Comparison of cause of death and hospital record diagnoses. Am J Public Health 69: 680–89. 1979.
- 25. Percy C, Stanek E, Gloeckler L. Accuracy of cancer death certificates and its effect on cancer mortality statistics. Am J Public Health 71:242–50. 1981.
- Kircher T, Nelson J, Burdo H. The autopsy as a measure of accuracy of the death certificate. N Engl J Med 313:1263–9. 1985.
- Michael S, Gard S, Schurman E, Kurth D. Sensitivity of death certificate data for monitoring diabetes mortality — Diabetic eye disease follow up study, 1985–1990. Morbidity and mortality weekly report 40(43):739–41. 1991.
- 28. National Center for Health Statistics. Vital statistics of the United States, 1991, vol II, mortality. Washington: Public Health Service. 1995.
- 29. National Center for Health Statistics. Vital statistics of the United States, 1992, vol II, mortality. Washington: Public Health Service. 1996.
- 30. Mason TJ. The development of the series of U.S. cancer atlases: Implications for future epidemiologic research. Stat Med 14:473–9. 1995.
- 31. Makuc DM, Haglund B, Ingram DD, et al. Health service areas for the United States. National Center for Health Statistics. Vital Health Stat 2(112). 1991.
- 32. Strategic Mapping, Inc. Atlas GIS. Santa Clara, California. 1992.
- 33. Dent BD. Cartography: Thematic map design. 3d ed. Dubuque, Iowa: Wm. C. Brown. p 56. 1993.
- 34. Sirken M, Herrmann D, White AA. Cognitive aspects of designing statistical maps.

- Proceedings of the survey research methods section of the 1993 annual meeting of the American Statistical Association. 586–91. 1994.
- 35. Pickle LW, Herrmann D, Kerwin J, et al. The impact of statistical graphic design on interpretation of disease rate maps.

 Proceedings of the statistical graphics section of the 1993 annual meeting of the American Statistical Association. 111–6. 1994.
- 36. Lewandowsky S, Herrmann DJ, Behrens JT, et al. Perception of clusters in statistical maps. Appl Cog Psych 7:533–51. 1993.
- 37. White AA, Pickle LW, Herrmann DJ, et al. Map design preferences associated with professional discipline. Proceedings of the statistical graphics section of the 1994 annual meeting of the American Statistical Association. 54–9. 1995.
- 38. Maher RJ. The interpretation of statistical maps as a function of the map reader's profession. In: Pickle LW, Herrmann DJ, eds. Cognitive aspects of statistical mapping. Working paper series report no 18. Hyattsville, Maryland: National Center for Health Statistics, 249–74, 1995.
- 39. Lewandowsky S, Behrens JT, Pickle LW, et al. Perception of clusters in mortality maps: Representing magnitude and statistical reliability. In: Pickle LW, Herrmann DJ, eds. Cognitive aspects of statistical mapping. Working paper series report no 18. Hyattsville, Maryland: National Center for Health Statistics. 107–32. 1995.
- 40. Pickle LW, Herrmann DJ, Wilson BF. A legendary study of statistical map reading: The cognitive effectiveness of statistical map legends. In: Pickle LW, Herrmann DJ, eds. Cognitive aspects of statistical mapping. Working paper series report no 18. Hyattsville, Maryland: National Center for Health Statistics. 233–48. 1995.
- 41. Hastie R, Hammerle O, Kerwin J, et al. Human performance reading statistical maps. J Exp Psych: Applied 2:3–16. 1996.
- 42. Lewandowsky S, Behrens JT. Accuracy of cluster detection in mortality maps. In: Pickle

- LW, Herrmann DJ, eds. Cognitive aspects of statistical mapping. Working paper series report no 18. Hyattsville, Maryland: National Center for Health Statistics. 133–48, 1995.
- 43. Carswell CM, Kinslow HS, Pickle LW, Herrmann DJ. Using color to represent magnitude in statistical maps: The case for double-ended scales. In: Pickle LW, Herrmann DJ, eds. Cognitive aspects of statistical mapping. Working paper series report no 18. Hyattsville, Maryland: National Center for Health Statistics. 201–28, 1995.
- 44. Brewer CA, MacEachren AM, Pickle LW. Evaluation of map color schemes for the NCHS mortality atlas. Proceedings of the international symposium on computer mapping in epidemiology and environmental health, Tampa, Florida. 1995. In press.
- 45. MacEachren AM, Brewer CA, Pickle LW. Mapping health statistics: Representing data reliability. Proceedings of the 17th international cartographic conference, Barcelona, Spain. September 3–9, 1995. Barcelona: Institut Cartographic de Catalunya. 311–9. 1995.
- 46. Carr D. Converting plots to tables. Technical report no 101. Center for Computational Statistics. Fairfax, Virginia: George Mason University. 1994.
- 47. Fleiss JL. Statistical methods for rates and proportions. 2d ed. New York: John Wiley and Sons. 1981.
- 48. Pickle LW, Mungiole M, Jones GK, White AA. Analysis of mapped mortality data by mixed effects models. Proceedings of the biometrics section of the 1996 annual meeting of the American Statistical Association. In press.
- 49. Chiang CL. Standard error of the age-adjusted death rate. Vital statistics—selected reports; vol 47 no 9. Washington: U.S. Government Printing Office. 1961.
- 50. Hansen KM. Headbanging: Robust smoothing in the plane. IEEE transactions on geoscience and remote sensing 29(3):369–78. 1991.

- 51. Kafadar K. Choosing among two-dimensional smoothers in practice. J Comp Simul 18: 419–39. 1994.
- 52. Mungiole M, Pickle LW, Simonson KH, White AA. Application of a weighted headbanging algorithm to mortality data maps. Proceedings of the statistical graphics section of the 1996 annual meeting of the American Statistical Association. In press.
- 53. National Center for Health Statistics. Health, United States, 1994. Hyattsville, Maryland: Public Health Service, 1995.
- 54. American Heart Association. Heart and stroke facts. 1996.
- 55. Gillum RF. Trends in acute myocardial infarction and coronary heart disease death in the United States. J Am Coll Cardiol 23:1273–7. 1993.
- 56. Pooling project research group. Relationship of blood pressure, serum cholesterol, smoking habit, relative weight and ECG abnormalities to incidence of major coronary events: Final report of the pooling project. J Chron Dis 31:201–306. 1978.
- 57. Gillum RF. Cardiovascular disease in the United States: Mortality, prevalence and incidence. In: Kapoor AS, Singh BN, eds. Prognosis and risk assessment in cardiovascular disease. New York: Churchill Livingstone. 49–59. 1993.
- 58. Anderson KM, Wilson PWF, Odell PM, Kannel WB. AHA statement: An updated coronary risk profile. Circulation 83(1):356–62. 1991.
- 59. Willett WC, Manson JE, Stampfer MJ, et al. Weight, weight change, and coronary heart disease in women. JAMA 273(6):461–5. 1995.
- 60. Ascherio A, Willett WC. New directions in dietary studies of coronary heart disease. J Nutr 125(3 supp):647S–655S. 1995.
- 61. Centers for Disease Control. Public health focus: Physical activity and the prevention of coronary heart disease. Morbidity and mortality weekly report 42(35):669–72. 1993.

- 62. Manson JE. Postmenopausal hormone therapy and atherosclerotic disease. Amer Heart J 128:1337–43. 1994.
- 63. Kannel WB, McGee D, Gordon T. A general cardiovascular risk profile: The Framingham study. Amer J Cardiology 38:46–51. 1976.
- 64. Gillum RF. Prevalence of cardiovascular and pulmonary diseases and risk factors by region and urbanization in the United States. J Natl Med Assoc 86:105–12. 1994.
- 65. Gillum RF. Geographic variation in sudden coronary death. Am Heart J 19(2):380–89. 1990.
- 66. Ingram D, Gillum RF. Regional and urbanization differentials in coronary heart disease mortality in the United States, 1968–85. J Clin Epidemiol 42(9):857–868. 1989.
- 67. Centers for Disease Control. Cardiovascular disease surveillance, ischemic heart disease, 1980–1989. 1993.
- 68. Feinleib M, Lentzner H, Collins J, et al. Regional variation in coronary heart disease mortality and morbidity. In: Higgins M, Luepker R, eds. Trends in coronary heart disease mortality. New York: Oxford University Press. 31–43. 1988.
- 69. Kosary CL, Ries LAG, Miller BA, et al., eds. SEER cancer statistics review, 1973–1992: Tables and graphs, National Cancer Institute. DHHS pub no (NIH) 96–2789. Washington: U.S. Government Printing Office. 1996.
- 70. Davis DL, Dinse GE, Hoel DG. Decreasing cardiovascular disease and increasing cancer among whites in the United States from 1973 through 1987. JAMA 271(6):431–37. 1994.
- 71. American Cancer Society, Inc. Cancer facts and figures 1994. Atlanta: American Cancer Society. 1994.
- 72. Kosary CL, Devesa SS. All cancer sites combined and all sites excluding lung and bronchus. In: Miller BA, Ries LAG, Hankey BF, et al., eds. SEER cancer statistics review, 1973–1990. Washington: U.S. Government Printing Office. II.1–II.51. 1993.

- 73. Olopade OI, Cummings S. Genetic counseling for cancer: Part I. Principles & practice of oncology updates 10(1):1–13. 1996.
- 74. Olopade OI, Cummings S. Genetic counseling for cancer: Part II. Principles & practice of oncology updates 10(2):1–11. 1996.
- 75. Ginsberg RJ, Kris MG, Armstrong JG. Chapter 23: Cancer of the lung. In: DeVita VT, Jr.; Hellman S; Rosenberg SA; eds. Cancer: Principles and practice of oncology. Philadelphia: J.B. Lippincott Company. 673–758. 1993.
- Ries LAG, Schatzkin A, Kaplan R. Colon and rectum. In: Miller BA, Ries LAG, Hankey BF, et al., eds. SEER cancer statistics review, 1973– 1990. Washington: U.S. Government Printing Office. VI.1–VI.22. 1993.
- 77. Cohen AM, Minsky BD, Schilsky RL. Chapter 30: Colon cancer. In: DeVita VT, Jr.; Hellman S; Rosenberg SA; eds. Cancer: Principles and practice of oncology. Philadelphia: J.B. Lippincott Company. 929–77. 1993.
- 78. Miller BA, Hayes RB, Potosky AL, et al. Prostate. In: Miller BA, Ries LAG, Hankey BF, et al., eds. SEER cancer statistics review, 1973–1990. Bethesda, Maryland: National Cancer Institute. Washington: U.S. Government Printing Office. XXII.1–XXII.15. 1993.
- 79. Hanks GE, Myers CE, Scardino PT. Chapter 35: Cancer of the prostate. In: DeVita VT, Jr.; Hellman S; Rosenberg SA; eds. Cancer: Principles and practice of oncology. Philadelphia: J.B. Lippincott Company. 1073–1113. 1993.
- 80. Morton RA. Racial differences in adenocarcinoma of the prostate in North American men. Urology 44(5):637–45. 1994.
- 81. Velentgas P, Daling JR. Risk factors for breast cancer in younger women. Monogr Natl Cancer Inst 16:15–22. 1994.
- 82. Nichols DH. The epidemiologic characteristics of breast cancer. Clin Obstet Gynecol 37(4): 925–30. 1994.

- 83. Harris JR, Morrow M, Bonadonna G. Chapter 40: Cancer of the breast. In: DeVita VT, Jr.; Hellman S; Rosenberg SA; eds. Cancer: Principles and practice of oncology. Philadelphia: J.B. Lippincott Company. 1264–1332. 1993.
- 84. Kelsey JL. Breast cancer epidemiology: Summary and future directions. Epi Rev 15(1):256–63. 1993.
- 85. Sturgeon SR, Schairer C, Gail M, et al. Geographic variation in mortality from breast cancer among white women in the United States. J Natl Cancer Inst 87:1846–53. 1995.
- 86. National Heart, Lung, and Blood Institute. Morbidity and mortality: Chartbook on cardiovascular, lung, and blood diseases. Bethesda, Maryland: National Institutes of Health 36–42. 1994.
- 87. Centers for Disease Control and Prevention. Cardiovascular disease surveillance, stroke, 1980–1989. 1994.
- 88. Kistler JP, Ropper AH, Martin JB.
 Cerebrovascular diseases. In: Isselbacher KJ,
 Martin JB, Braunwald E, et al., eds. Harrison's
 principles of internal medicine. New York:
 McGraw-Hill, Inc. 2233–56. 1994.
- 89. Dyken ML, Wolf PA, Barnett HJM, et al. News from the American Heart Association: Risk factors in stroke. A statement for physicians by the Subcommittee on Risk Factors and Stroke of the Stroke Council. Stroke 15(6):1105–11. 1984.
- 90. Ostfeld AM, Wilk E. Epidemiology of stroke, 1980–1990: A progress report. Epi Rev 12: 253–6. 1990.
- 91. Casper ML, Wing S, Anda RF, Knowles M, Pollard RA. The shifting stroke belt: Changes in the geographic pattern of stroke mortality in the United States, 1962–1988. Stroke 26:755–60. 1995.
- 92. Lanska DJ, Peterson PM. Geographic variation in the decline of stroke mortality in the United States. Stroke 26:1159–65. 1995.

- 93. Wing S, Casper M, Davis WB, et al. Stroke mortality maps: United States whites aged 35–74 years, 1962–1982. Stroke 19:1507–13. 1988.
- 94. Lanska DJ, Peterson PM. Effects of interstate migration on the geographic distribution of stroke mortality in the United States. Stroke 26:554–61. 1995.
- 95. Baker SP, O'Neill B, Ginsburg MJ, Li G. The injury fact book. New York: Oxford University Press. 1992.
- 96. National Center for Environmental Health and Injury Control (NCEHIC). Position papers from the Third National Injury Control Conference. Washington: U.S. Government Printing Office. 261–96, 329–37. 1992.
- 97. Fingerhut LA, Jones C, Makuc DM. Firearm and motor vehicle injury mortality; variations by State, race, and ethnicity: United States 1990–1991. Advance data from vital and health statistics; no 242. Hyattsville, Maryland: National Center for Health Statistics. 1994.
- 98. Bates DV. Respiratory function in disease. Philadelphia: W.B. Saunders Company. 1989.
- 99. Cherniack NS. Chronic obstructive pulmonary disease. Philadelphia: W.B. Saunders Company. 227–32. 1991.
- Centers for Disease Control. Update: Influenza activity United States, 1991–1992 season.
 Morbidity and mortality weekly report 41(04): 63–5. 1992.
- 101. Centers for Disease Control. Update: Influenza activity United States, 1992–1993 season.

 Morbidity and mortality weekly report 42(20):385–87. 1993.
- 102. LaForce FM, Nichol KL, Cox NJ. Influenza: virology, epidemiology, disease, and prevention. Am J Prev Med 10(supp):31–44. 1994.
- 103. Betts RF. Influenza virus. In: Mandell GL, Bennett JE, Dolin R, eds. Principles and practice of infectious diseases. New York: Churchill Livingstone. 1546–67. 1995.

- 104. Donowitz GR, Mandell GL. Acute pneumonia. In: Mandell GL, Bennett JE, Dolin R, eds. Principles and practice of infectious diseases. New York: Churchill Livingstone. 619–36. 1995.
- 105. Flegal KM, Ezzati TM, Harris MI, et al. Prevalence of diabetes in Mexican Americans, Cubans, and Puerto Ricans from the Hispanic Health and Nutrition Examination Survey, 1982– 1984. Diabetes care 14(supp 3):628–38. 1991.
- 106. Lernmark A. Insulin-dependent diabetes mellitus. In: Davidson JK, ed. Clinical diabetes mellitus: A problem-oriented approach. New York: Theime Medical Publishers, Inc. 35–49. 1991.
- 107. Pareschi PL, Tomasi F. Epidemiology of diabetes mellitus. In: Morsiani M, ed. Epidemiology and screening of diabetes. Boca Raton, Florida: CRC Press, Inc. 77–114. 1989.
- 108. Dahlquist G. Non-genetic risk determinants of type 1 diabetes. Diabete Metab 20:251–57. 1994.
- 109. Centers for Disease Control and Prevention. Suicide among older persons United States, 1980–1992. Morbidity and mortality weekly report 45(1):3–6. 1996.
- 110. O'Carroll PW, Rosenberg M, Mercy JA. Suicide. In: Rosenberg ML, Fenley MA, eds. Violence in America: A public health approach. New York: Oxford University Press. 184–96. 1991.
- 111. Monk M. Epidemiology of suicide. Epi Reviews 9:51–69. 1987.
- 112. Brent DA, Perper JA, Allman CJ, et al. The presence and accessibility of firearms in the homes of adolescent suicides. JAMA 266(21):2989–95. 1991.
- 113. Fingerhut LA. Firearm mortality among children, youth, and young adults 1–34 years of age, trends and current status: United States, 1985–1990. Advance data from vital and health statistics; no 231. Hyattsville, Maryland: National Center for Health Statistics. 1993.
- 114. National Center for Health Statistics. Excess deaths and other mortality measures for the Hispanic population. 1994.

- 115. Centers for Disease Control and Prevention. Deaths and hospitalizations from chronic liver disease and cirrhosis - United States, 1980– 1989. Morbidity and mortality weekly report 41(52):969–73. 1993.
- 116. Centers for Disease Control. Chronic disease reports: Deaths from chronic liver disease United States, 1986. Morbidity and mortality weekly report 38(46):792, 797–9. 1989.
- 117. Corrao G, Lepore AR, Torchio P, et al. Interaction between dietary pattern and alcohol intake on the risk of liver cirrhosis. Rev Epidemiol Sante Publique 43:7–17. 1995.
- 118. Boyer JL, Reuben A. Chronic hepatitis. In: Schiff L, Schiff ER, eds. Diseases of the liver, volume 1. Philadelphia: J.B. Lippincott Company. 586–637. 1993.
- 119. Centers for Disease Control and Prevention. Update: Mortality attributable to HIV infection among persons aged 25–44 years - United States, 1991 and 1992. Morbidity and mortality weekly report 42(45):869–72. 1993.
- 120. Centers for Disease Control and Prevention. HIV/AIDS surveillance report, 1994 6(2):30–1. 34. 1994.
- 121. Auerbach JD, Wypijewska C, Brodie HKH, eds. AIDS and behavior: An integrated approach. Washington: National Academy Press. 47–77. 1994.
- 122. Holmberg SD, Curran JW. The epidemiology of HIV infection in industrialized countries. In: Holmes KK, Mardh P, Sparling PF, Wiesner PJ, eds. Sexually transmitted diseases. New York: McGraw-Hill Information Services Company. 343–53. 1990.
- 123. Centers for Disease Control and Prevention. HIV/AIDS surveillance report, 1993 5(4):1. 1993.
- 124. Centers for Disease Control and Prevention. Trends in rates of homicide United States, 1985–1994. Morbidity and mortality weekly report 45(22):460–4. 1996.

- 125. Rosenberg M, Mercy JA. Assaultive violence. In: Rosenberg ML, Fenley MA, eds. Violence in America: A public health approach. New York: Oxford University Press. 14–50. 1991.
- 126. Centers for Disease Control. Homicide among young black males United States, 1978–1987. Morbidity and mortality weekly report 39(48):869–73. 1990.
- 127. Brillinger DR. The natural variability of vital rates and associated statistics. Biometrics 42:693–712. 1986.
- 128. Pickle LW. Measuring the dispersion of cancer mortality rates by the interquartile range. In: Page C, LePage R, eds. Computing science and statistics. Statistics of many parameters: Curves, images, spatial models. New York: Springer-Verlag. 496–500. 1992.
- 129. Manton KG, Woodbury MA, Stallard E, et al. Empirical Bayes procedures for stabilizing maps of U.S. cancer mortality rates. JASA 84:637–50. 1989.

- 130. Clayton D, Kaldor J. Empirical Bayes estimates of age-standardized relative risks for use in disease mapping. Biometrics 43:671–81. 1987.
- 131. Pickle LW, White AA. Effects of the choice of age-adjustment method on maps of death rates. Stat Med 14:615–27. 1995.
- 132. Wolfinger R, O'Connell M. Generalized linear mixed models: A pseudo-likelihood approach. J Stat Comp Simul 48:233–43. 1993.
- 133. Royall R. Model robust confidence intervals using maximum likelihood estimators. Int Stat Rev 54:221–6. 1986.
- 134. McCullagh P, Nelder JA. Generalized linear models. 2d ed. New York: Chapman and Hall. 1989.
- 135. Siegel PZ, Frazier EL, Mariolis P, et al.
 Behavioral risk factor surveillance, 1991:
 Monitoring progress toward the Nation's Year
 2000 health objectives. In: CDC surveillance
 summaries, August 1993. Morbidity and
 mortality weekly report 42(SS-4):1-17. 1993.

Health Service Areas (HSA's) are aggregations of counties and independent cities based on a cluster analysis of where Medicare patients obtained routine hospital care in 1988 (31) (See fold-out map.). The clustering algorithm defined the HSA as an area within which its residents were more likely to seek hospital care than to travel outside it. The 805 HSA's used for this atlas differ from the original 802 HSA definitions (referred to as the "800-unlinked solution" by Makuc et al. (31)). These differences are as follows.

 HSA's for Alaska and Hawaii were created using the same clustering algorithm. The counties (or territories) included for these HSA's are the following.

817: Ketchikan Gateway, Wrangell - Petersburg, Prince of Wales - Outer Ketchikan, AK

818: Juneau, Sitka, Angoon, Haines, Skagway-Yakutat. AK

819: Fairbanks North Star, Southeast Fairbanks, Upper Yukon, Yukon - Koyukuk, AK

820: Remainder of AK

821: Honolulu, Maui, Kalawao, HI

822: Kauai, HI 823: Hawaii, HI

- The islands of Nantucket, Massachusetts (HSA 111) and Duke Island, Massachusetts (HSA 120) were combined with Boston, Massachusetts (HSA 22) and Rockland, New York (HSA 133) was grouped with Bergen City, New Jersey (HSA 36) to achieve a minimum HSA size of 250 square miles for visibility on the maps. Only New York City (HSA 94) remains below this minimum. It is shown at larger scale east of its actual location and is labeled "NYC" on the maps.
- New York City (HSA 94) was redefined to be consistent with its census designation. From the original Series Report definitions, Kings and Richmond counties, New York (HSA 113) and Queens, New York (removed from HSA 83) were added to HSA 94.

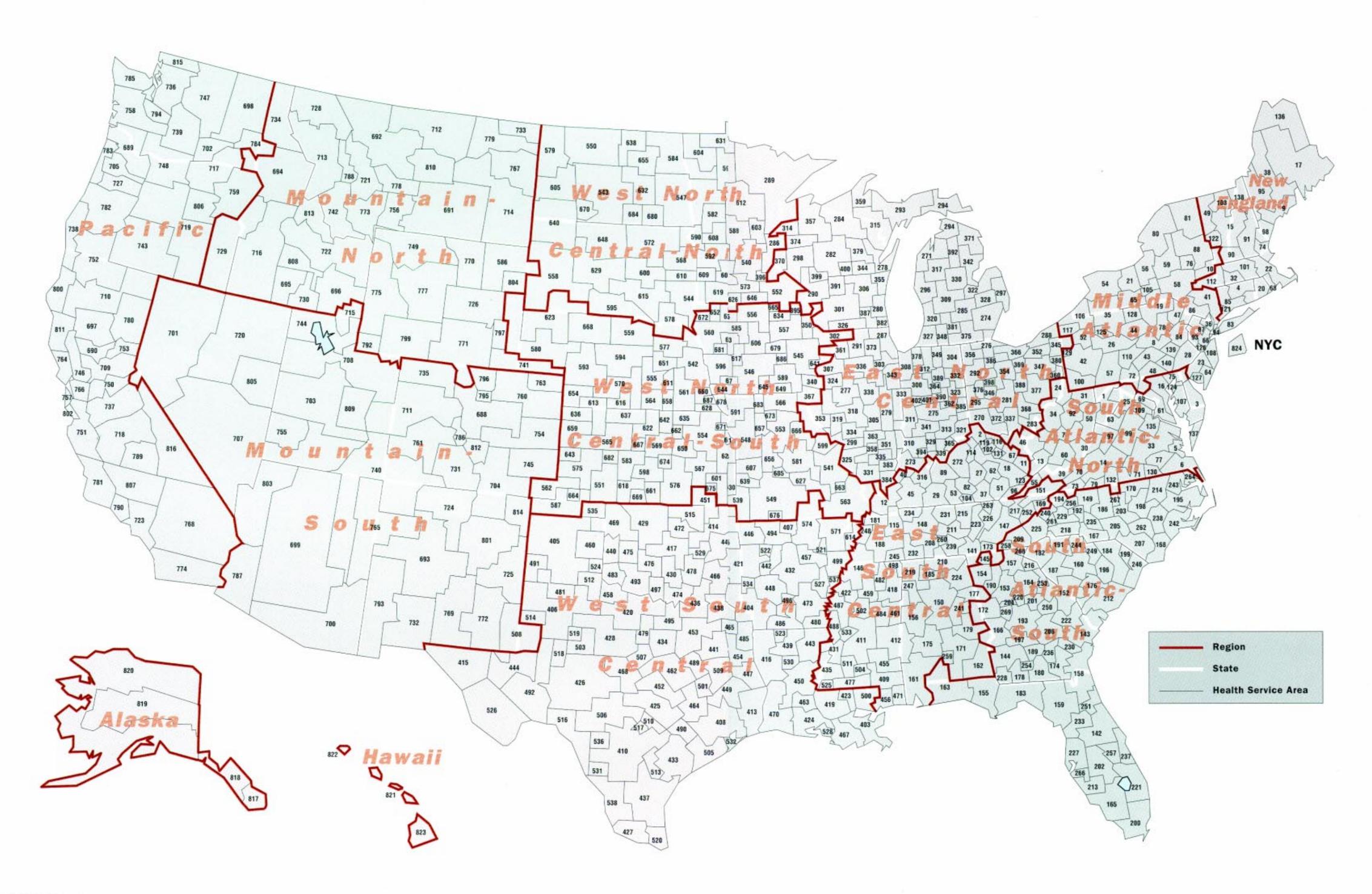
Names have been developed for the 805 HSA's to assist readers in recognizing their geographic location (DD Ingram, personal communication). The HSA's were originally identified only by numbers. The primary identification of the HSA's is by county name because counties were used in the creation of the HSA's. For some HSA's a place name (for a city, town, or other Census Designated Place (CDP)) was also

chosen for use in the HSA name because it may be better known to readers than the county name. Therefore, to meet the objective of recognizability, the following approach was taken to create the HSA names.

- A maximum of two county names was used in an HSA name. For HSA's comprised of only one county, the name of that county appears in the HSA name. For HSA's comprised of two counties or more, the county with the largest number of Medicare hospital stays in 1988 (primary county) appears first in the HSA name and the county with the second largest number of hospital stays (secondary county) appears second. The twoletter State abbreviation was included with each county name as some HSA's include counties from more than one State.
- If the primary or secondary county in the HSA was part of a Metropolitan Statistical Area as defined by the U.S. Bureau of the Census in 1990, a place name (for a city, town, CDP, etc.) was included in the HSA name. The place name used in the HSA name was the name of the place with the largest 1990 population from either the primary or secondary county in the HSA. The place name appears in the HSA name in parentheses, following either the primary or secondary county name.
 - To avoid redundancy, place names were not included in the names of five HSA's for which the county was actually an independent city. These HSA's are Washington, D.C.-Montgomery, Maryland (HSA 61); St. Louis, Missouri-St. Louis city, Missouri (HSA 541); Newport News city, Virginia -Hampton city, Virginia (HSA 5); and Norfolk/Portsmouth city, Virginia-Virginia Beach city, Virginia (HSA 6).
 - A place name was not included in the HSA name for Nassau, New York-Suffolk, New York (HSA 83), Washington, Pennsylvania-Fayette, Pennsylvania (HSA 100), or St. Croix, Wisconsin-Goodhue, Minnesota (HSA 370) because their primary and secondary counties include a number of small communities that are suburbs of a large metropolitan area. It was not possible to select one of them as the principal place in the county.

 A place name was not included in the HSA name if the primary or secondary county in the HSA was not part of a Metropolitan Statistical Area. For these HSA's, the population of the largest place tended to be small or the county was composed of numerous small towns with similar population size.

BOUNDARIES FOR REGIONS, STATES, AND HEALTH SERVICE AREAS



SOURCE: CDC/NCHS

	Number			Number	
HSA	of	. HSA name	HSA	of	. HSA name
number	counties in HSA	пол паше	number	counties in HSA	S HAITIE
1	5	Allegany (Cumberland), MD - Garrett, MD	52	3	Venango, PA - Clarion, PA
2	6	Kenton (Covington), KY - Campbell, KY	53	4	Barren, KY - Monroe, KY
3	5	Sussex, DE - Wicomico, MD	54	7	Erie (Buffalo), NY - Monroe, NY
4	3	Hartford (Hartford), CT - Windham, CT	55	3	Wise, VA - Lee, VA
5	6	Newport News city, VA - Hampton city, VA	56	3	Onondaga (Syracuse), NY - Cayuga, NY
6	7	Norfolk/Portsmouth city, VA - Virginia	57	4	Cambria, PA - Blair (Altoona), PA
		Beach city, VA	58	4	Otsego, NY - Delaware, NY
7	6	Kanawha (Charleston), WV - Putnam, WV	59	3	Oneida (Utica), NY - Herkimer, NY
8	5	Schuylkill, PA - Montour, PA	60	4	Raleigh, WV - Fayette, WV
9	4	Cumberland (Portland), ME - Knox, ME	61	5	Washington, DC - Montgomery, MD
10	3	Albany (Albany), NY - Rensselaer, NY	62	4	Madison, KY - Rockcastle, KY
11	3	Floyd, KY - Johnson, KY	63	3	Rockingham, VA - Page, VA
12	11	McCracken, KY - Graves, KY	64	2	Atlantic (Atlantic City), NJ - Cape May, NJ
13	3	Pike, KY - Logan, WV	65	3	Steuben, NY - Chemung (Elmira), NY
14	9	Roanoke (Roanoke), VA - Campbell, VA	66	4	Essex (Newark), NJ - Union, NJ
15	5	Grafton, NH - Washington, VT	67	4	Rowan, KY - Morgan, KY
16	5	Baltimore (Baltimore), MD - Anne	68	2	Bristol (New Bedford), MA - Newport, RI
		Arundel, MD	69	4	Alexandria city, VA - Arlington -
17	4	Penobscot (Bangor), ME - Hancock, ME			Alexandria (Arlington), VA
18	13	Fayette (Lexington - Fayette), KY - Perry,	70	4	Montgomery, VA - Pulaski, VA
		KY	71	3	Halifax, VA - Mecklenburg, VA
19 20	5 5	Broome (Binghamton), NY - Bradford, PA Providence (Providence), RI - New	72	3	Franklin, PA - Washington (Hagerstown), MD
		London, CT	73	3	Grayson, VA - Alleghany, NC
21	4	Ontario (Geneva), NY - Wayne, NY	74	2	Essex (Lynn), MA - Rockingham, NH
22	7	Middlesex, MA - Suffolk (Boston), MA	75	3	New Castle (Wilmington), DE - Harford, MD
23	4	Camden (Camden), NJ - Burlington, NJ	76	2	Montgomery (Amsterdam), NY - Fulton, NY
24	6	Ohio (Wheeling), WV - Belmont, OH	77	4	Dinwiddie (Petersburg), VA - Prince
25	8	Frederick, VA - Berkeley, WV	70	0	George, VA
26	3	Clearfield, PA - Centre (State College), PA	78	3	Luzerne (Wilkes - Barre), PA - Columbia, PA
27	5	Boyle, KY - Lincoln, KY	79	2	Henry, VA - Patrick, VA
28	5	Philadelphia (Philadelphia), PA	80	3	St. Lawrence, NY - Jefferson, NY
00	0	Montgomery, PA	81	3	Clinton, NY - Franklin, NY
29	6	Warren, KY - Logan, KY	82	4	Taylor, KY - Russell, KY
30	4	Alleghany, VA - Greenbrier, WV	83	2	Nassau, NY - Suffolk, NY
31	4	Monongalia, WV - Marion, WV	84	4	Lehigh (Allentown), PA - Northampton, PA
32 33	2 20	Hampden (Springfield), MA - Hampshire, MA Henrico (Richmond), VA - Prince Edward,	85	3	New Haven (New Haven), CT - Litchfield, CT
24	0	VA	86 87	3	Orange (Newburgh), NY - Sullivan, NY
34	8	Wood (Parkersburg), WV - Washington, OH	87	2	Morris (Parsippany - Troy Hills Township),
35	5	Cattaraugus, NY - McKean, PA	00	E	NJ - Sussex, NJ
36 37	4 3	Bergen, NJ - Hudson (Jersey City), NJ Pulaski, KY - Wayne, KY	88	5	Schenectady (Schenectady), NY - Warren, NY
38	3	Kennebec, ME - Somerset, ME	89	5	Hardin, KY - Grayson, KY
39	5	Mercer, WV - Tazewell, VA	90	2	Cheshire, NH - Windham, VT
40	2	Henderson (Henderson), KY - Union, KY	91	4	Hillsborough (Manchester), NH -
41	4	Westchester (Yonkers), NY - Dutchess, NY			Merrimack, NH
42	6	Allegheny (Pittsburgh), PA -	92	6	Harrison, WV - Lewis, WV
		Westmoreland, PA	93	2	Warren (Phillipsburg), NJ - Hunterdon, NJ
43	4	Dauphin (Harrisburg), PA - Cumberland, PA	94	5	New York, NY - Kings (New York), NY
44	2	Lycoming (Williamsport), PA - Clinton, PA	95	3	Androscoggin (Lewiston), ME - Oxford, ME
45	7	Hopkins, KY - Christian (Hopkinsville), KY	96	3	Bell, KY - Claiborne, TN
46	9	Cabell (Huntington), WV - Boyd, KY	97	3	Augusta, VA - Rockbridge, VA
47	2	Lackawanna (Scranton), PA - Wayne, PA	98	2	York, ME - Strafford (Rochester), NH
48	3	York (York), PA - Frederick, MD	99	10	Albemarle (Charlottesville), VA -
49	4	Chittenden (Burlington), VT - Franklin, VT			Culpeper, VA
50	4	Randolph, WV - Barbour, WV	100	3	Washington, PA - Fayette, PA
50	•				

	Number			Number	
HSA	of	USA nama	HSA	of	USA nama
number	counties in HSA	HSA name	number	counties in HSA	HSA name
102	2	Bourbon (Paris), KY - Nicholas, KY	164	8	Clarke (Athens), GA - Barrow, GA
103	3	Orleans, VT - Caledonia, VT	165	4	Lee (Cape Coral), FL - Collier, FL
104	2	Cumberland, KY - Clinton, KY	166	9	Muscogee (Columbus), GA - Russell, AL
105	2	Cortland, NY - Tompkins, NY	167	3	Mecklenburg (Charlotte), NC - Union, NC
106	3	Erie (Erie), PA - Chautauqua, NY	168	3	New Hanover (Wilmington), NC -
107	4	Talbot, MD - Dorchester, MD	160	2	Brunswick, NC
108 109	2 2	Ocean (Brick Township), NJ - Monmouth, NJ Prince William (Dale City), VA - Fauquier,	169 170	3 5	Washington (Johnson City), TN - Carter, TN Durham (Durham), NC - Vance, NC
109	2	VA	171	7	Montgomery (Montgomery), AL -
110	3	Mifflin, PA - Huntingdon, PA	111	,	Covington, AL
112	3	Berkshire (Pittsfield), MA - Columbia, NY	172	6	Troup (La Grange), GA - Coweta, GA
114	3	Franklin, KY - Owen, KY	173	4	Bradley, TN - Gilmer, GA
115	4	Calloway, KY - Carroll, TN	174	4	Ware, GA - Bacon, GA
116	3	Mason, KY - Fleming, KY	175	4	Dallas, AL - Marengo, AL
117	1	Crawford, PA	176	4	Orangeburg, SC - Bamberg, SC
119	2	Harrison, KY - Robertson, KY	177	4	Calhoun (Anniston), AL - Carroll, GA
121	1	Fairfield (Bridgeport), CT	178	3	Thomas, GA - Grady, GA
122	1	Rutland, VT	179	4	Lee (Auburn), AL - Elmore, AL
123	1	Letcher, KY	180	6	Lowndes, GA - Cook, GA
124	1	Kent, MD	181	3	Obion, TN - Weakley, TN
125	1	Elk, PA	182	4	Greenville (Greenville), SC - Anderson, SC
126	1	Mercer (Trenton), NJ	183	8	Leon (Tallahassee), FL - Madison, FL
127	1	Cumberland (Vineland), NJ	184	5	Florence (Florence), SC - Darlington, SC
128	1	Tioga, PA	185	2	Morgan (Decatur), AL - Lawrence, AL
129	1	Lawrence, PA	186	4	Guilford (Greensboro), NC -Rockingham,
130	2	Greensville, VA - Brunswick, VA	107	5	NC
131 132	2 2	Montgomery, KY - Bath, KY Pittsylvania (Danville), VA - Caswell, NC	187 188	5 8	Greenwood, SC - Laurens, SC Madison (Jackson), TN - Gibson, TN
135	4	Spotsylvania, VA - Caroline, VA	189	4	Tift, GA - Ben Hill, GA
136	1	Aroostook, ME	190	5	Cobb (Marietta), GA - Cherokee, GA
137	2	Northampton, VA - Accomack, VA	191	4	Spartanburg (Spartanburg), SC - Union, SC
138	2	Coos, NH - Essex, VT	192	2	Iredell (Statesville), NC - Alexander, NC
139	1	Berks (Reading), PA	193	11	Bibb (Macon), GA - Houston, GA
140	1	Lancaster (Lancaster), PA	194	3	Watauga, NC - Ashe, NC
141	6	Hamilton (Chattanooga), TN - Catoosa, GA	195	6	Pitt, NC - Beaufort, NC
142	6	Orange (Orlando), FL - Volusia, FL	196	3	Sumter, SC - Clarendon, SC
143	9	Chatham (Savannah), GA - Beaufort, SC	197	5	Sumter, GA - Crisp, GA
144	9	Dougherty (Albany), GA - Early, GA	198	4	Wake (Raleigh), NC - Wilson, NC
145	2	Whitfield, GA - Murray, GA	199	2	Marion, SC - Dillon, SC
146	9	Shelby (Memphis), TN - Panola, MS	200	3	Dade (Miami), FL - Broward, FL
147	8	Knox (Knoxville), TN - Blount, TN	201	3	Baldwin, GA - Putnam, GA
148	7	Davidson (Nashville - Davidson), TN -	202	3	Polk (Lakeland), FL - Highlands, FL
1.10	_	Dickson, TN	203	4	Orange (Chapel Hill), NC - Harnett, NC
149	5	Forsyth (Winston - Salem), NC - Surry, NC	204	2	Spalding (Griffin), GA - Butts, GA
150 151	8 6	Jefferson (Birmingham), AL - Walker, AL Sullivan (Kingsport), TN - Washington, VA	205 206	4 8	Moore, NC - Richmond, NC Laurens, GA - Dodge, GA
151	12	Richmond (South Augusta), GA - Aiken, SC	207	4	Robeson, NC - Columbus, NC
153	9	Fulton (Atlanta), GA - DeKalb, GA	207	4	Maury, TN - Giles, TN
154	5	Floyd, GA - Bartow, GA	209	4	Macon, NC - Jackson, NC
155	6	Bay (Panama City), FL - Jackson, FL	210	4	Madison (Huntsville), AL - Jackson, AL
156	4	Tuscaloosa (Tuscaloosa), AL - Pickens, AL	211	5	Rutherford (Murfreesboro), TN - Warren, TN
157	8	Hall, GA - Union, GA	212	5	Charleston (Charleston), SC - Colleton, SC
158	9	Duval (Jacksonville), FL - Glynn, GA	213	3	Sarasota (Sarasota), FL - Charlotte, FL
159	10	Alachua (Gainesville), FL - Columbia, FL	214	4	Edgecombe, NC - Halifax, NC
160	4	Richland (Columbia), SC - Lexington, SC	215	6	Putnam, TN - Overton, TN
161	5	Mobile (Mobile), AL - Baldwin, AL	216	3	Stephens, GA - Franklin, GA
162	7	Houston (Dothan), AL - Coffee, AL	217	4	Hamblen (Morristown), TN - Jefferson, TN
			218	4	Gaston (Gastonia), NC - Cleveland, NC

	Number			Number	
HSA	of	. HSA name	HSA	of	. HSA name
number	counties in HSA	TIOA Hame	number	counties in HSA	S Hon hame
219	4	Lauderdale (Florence), AL - Colbert, AL	277	5	Peoria (Peoria), IL - Tazewell, IL
220	2	Newton (Covington), GA - Jasper, GA	278	4	Brown (Green Bay), WI - Door, WI
221	4	Palm Beach (West Palm Beach), FL -	279	5	Champaign (Champaign), IL - Coles, IL
000	4	St. Lucie, FL	280	4	Milwaukee (Milwaukee), WI - Waukesha,
222	4	Bulloch, GA - Emanuel, GA	004	_	WI
223 224	3 4	Cumberland, TN - Fentress, TN	281 282	5	Franklin (Columbus), OH - Delaware, OH Wood, WI - Marathon (Wausau), WI
225	5	Etowah (Gadsden), AL - Marshall, AL Buncombe (Asheville), NC - Henderson, NC	283	5 4	Gallia, OH - Mason, WV
226	5	Anderson (Oak Ridge), TN - Roane, TN	284	6	Oneida, WI - Gogebic, MI
227	4	Pinellas, FL - Hillsborough (Tampa), FL	285	4	Ingham (Lansing), MI - Shiawassee, MI
228	3	Decatur, GA - Seminole, GA	286	5	Ramsey (St. Paul), MN - Polk, WI
229	3	Catawba (Hickory), NC - Burke, NC	287	4	Cook (Chicago), IL - Du Page, IL
230	2	Wayne, GA - Appling, GA	288	4	Cuyahoga (Cleveland), OH - Lake, OH
231	5	Sumner (Hendersonville), TN - Wilson, TN	289	8	St. Louis (Duluth), MN - Douglas, WI
232	3	Lawrence, TN - Wayne, TN	290	6	La Crosse (La Crosse), WI - Monroe, WI
233	2	Marion (Ocala), FL - Citrus, FL	291	4	Winnebago (Rockford), IL - Lee, IL
234	3	Montgomery (Clarksville), TN - Houston, TN	292	6	Allen (Lima), OH - Mercer, OH
235	3	Cabarrus (Concord), NC - Rowan, NC	293	5	Marquette, MI - Delta, MI
236	3	Coffee, GA - Jeff Davis, GA	294	5	Emmet, MI - Cheboygan, MI
237	2	Brevard (Palm Bay), FL - Indian River, FL	295	4	Montgomery (Dayton), OH - Greene, OH
238 239	4	Lenoir, NC - Wayne, NC	296 297	4	Muskegon (Muskegon), MI - Mason, MI
239 240	4 3	Coffee, TN - Franklin, TN Avery, NC - Mitchell, NC	297 298	2 5	St. Clair (Port Huron), MI - Sanilac, MI Eau Claire (Eau Claire), WI - Chippewa, WI
241	3	Talladega, AL - Clay, AL	299	4	Madison (Alton), IL - Jersey, IL
242	5	Craven, NC - Carteret, NC	300	5	Tippecanoe (Lafayette), IN - Clinton, IN
243	4	Hertford, NC - Chowan, NC	301	7	Dane (Madison), WI - Sauk, WI
244	2	York (Rock Hill), SC - Chester, SC	302	4	Dubuque (Dubuque), IA - Stephenson, IL
245	2	Hardin, TN - McNairy, TN	303	2	Will (Joliet), IL - Grundy, IL
246	2	Horry, SC - Georgetown, SC	304	6	Allen (Fort Wayne), IN - Whitley, IN
247	2	Marion, AL - Fayette, AL	305	4	Macon (Decatur), IL - Shelby, IL
248	3	Dyer, TN - Lauderdale, TN	306	5	Winnebago (Oshkosh), WI - Fond du Lac,
249	2	Kershaw, SC - Lancaster, SC			WI
250	2	Washington, GA - Jefferson, GA	307	4	Rock Island (Moline), IL - Henry, IL
251	2	St. Johns (St. Augustine), FL - Putnam, FL	308	4	Lake (Gary), IN - Porter, IN
252	2	Greene, TN - Cocke, TN	309	5	Kent (Grand Rapids), MI - Montcalm, MI
253	2	Elbert, GA - Wilkes, GA	310	6	Knox, IN - Daviess, IN
254 256	1	Colquitt, GA	311 312	6 4	Vigo (Terre Haute), IN - Sullivan, IN
257	1 1	Wilkes, NC Osceola (Kissimmee), FL	313	4	St. Joseph (South Bend), IN - Marshall, IN Bartholomew, IN - Jackson, IN
258	2	Cherokee, NC - Clay, NC	313	2	Washburn, WI - Burnett, WI
259	1	Butler, AL	315	5	Dickinson, MI - Marinette, WI
260	1	Bedford, TN	316	5	Daviess (Owensboro), KY - Ohio, KY
261	1	McDowell, NC	317	4	Wexford, MI - Osceola, MI
262	2	Cumberland (Fayetteville), NC - Sampson, NC	318	8	Sangamon (Springfield), IL - McDonough, IL
263	1	Scott, TN	319	3	Morgan, IL - Greene, IL
264	4	Pasquotank, NC - Camden, NC	320	2	Ottawa (Holland), MI - Allegan, MI
266	1	Manatee (Bradenton), FL	321	7	Jefferson (Madison), IN - Dearborn, IN
267	1	Alamance (Burlington), NC	322	3	Saginaw (Saginaw), MI - Huron, MI
268	1	Rabun, GA	323	3	Delaware (Muncie), IN - Randolph, IN
269	3	Upson, GA - Lamar, GA	324	2	Knox, IL - Warren, IL
270	7	Hamilton (Cincinnati), OH - Butler, OH	325	4	St. Clair (Belleville), IL - Randolph, IL
271	6	Grand Traverse, MI - Manistee, MI	326	3	Rock (Janesville), WI - Green, WI
272	9	Jefferson (Louisville), KY - Shelby, KY	327	3	Berrien (Benton Harbor), MI - Van Buren,
273 274	6 5	Vanderburgh (Evansville), IN - Gibson, IN	270	2	MI Genesee (Flint) ML-Langer MI
274 275	5 7	Wayne (Detroit), MI - Oakland, MI Marion (Indianapolis), IN - Hendricks, IN	328 329	2 2	Genesee (Flint), MI - Lapeer, MI Lawrence, IN - Orange, IN
275 276	, 5	Lucas (Toledo), OH - Monroe, MI	330	4	Midland (Midland), MI - Isabella, MI
210	ິ	Lucas (Toleuo), On - Mollioe, Mi	330	4	iviiuialiu (iviiuialiu), ivii - ISabella, ivii

	Number			Number	
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331	4	Williamson, IL - Jackson, IL	387	2	Jefferson, WI - Walworth, WI
332	4	Wells, IN - Jay, IN	388	2	Licking (Newark), OH - Knox, OH
333	4	Vermilion, IL - Edgar, IL	389	3	Grant, IN - Wabash, IN
334	2	Montgomery, IL - Macoupin, IL	390	2	Madison (Anderson), IN - Hamilton, IN
335	2	Jefferson, IL - Wayne, IL	391	2	Juneau, WI - Adams, WI
336	4	La Salle, IL - Bureau, IL	392	3	Crawford, MI - Otsego, MI
337	4	Scioto, OH - Ross, OH	394	1	Dubois, IN
338	3	McLean (Bloomington), IL - De Witt, IL	395	1	Winneshiek, IA
339	4	Floyd (New Albany), IN - Harrison, IN	396	1	Rice, MN
340	3	Des Moines, IA - Henry, IA	398	1	Logan, OH
341	4	Monroe (Bloomington), IN - Greene, IN	399	1	Jackson, WI
342	5	Bay (Bay City), MI - Ogemaw, MI	400	1	Portage, WI
343	2	Kankakee (Kankakee), IL - Iroquois, IL	401	1	Boone (Lebanon), IN
344	2	Outagamie (Appleton), WI - Waupaca, WI	402	1	Montgomery, IN
345	3	Mahoning (Youngstown), OH - Mercer, PA	403	7	Orleans (New Orleans), LA - Jefferson, LA
346	2	Clark (Springfield), OH - Champaign, OH	404	9	Bowie (Texarkana), TX - Miller, AR
347	5	Stark (Canton), OH - Tuscarawas, OH	405	15	Potter (Amarillo), TX - Moore, TX
348	2	Kalamazoo (Kalamazoo), MI - St. Joseph,	406	13	Lubbock (Lubbock), TX - Lamb, TX
		MI	407	2	Baxter, AR - Marion, AR
349	2	Elkhart (Elkhart), IN - Kosciusko, IN	408	8	Harris (Houston), TX - Montgomery, TX
350	3	Crawford, WI - Clayton, IA	409	5	Forrest, MS - Covington, MS
351	4	Richland, IL - Clay, IL	410	12	Bexar (San Antonio), TX - Guadalupe, TX
352	3	Summit (Akron), OH - Medina, OH	411	8	Hinds (Jackson), MS - Rankin, MS
353	6	Adams, IL - Marion, Mo	412	7	Lauderdale, MS - Newton, MS
354	5	Marion, OH - Crawford, OH	413	6	Jefferson (Beaumont), TX - Orange, TX
355	3	Sheboygan (Sheboygan), WI - Manitowoc,	414	6	Tulsa (Tulsa), OK - Creek, OK
		WI	415	4	El Paso (El Paso), TX - Culberson, TX
356	4	Williams, OH - Defiance, OH	416	7	Caddo (Shreveport), LA - Webster, LA
357	4	Ashland, WI - Price, WI	417	5	Oklahoma (Oklahoma City), OK -
358	2	Marion, IL - Washington, IL			Pottawatomie, OK
359	3	Houghton, MI - Baraga, MI	418	5	Lee, MS - Prentiss, MS
360	2	Jefferson (Steubenville), OH - Harrison, OH	419	8	East Baton Rouge (Baton Rouge), LA -
361	3	Clinton, IA - Whiteside, IL			Ascension, LA
362	2	Henry (New Castle), IN - Hancock, IN	420	8	Wichita (Wichita Falls), TX - Young, TX
363	2	Effingham, IL - Fayette, IL	421	8	Sebastian (Fort Smith), AR - Crawford, AR
364	3	Howard (Kokomo), IN - Cass, IN	422	3	Coahoma, MS - Quitman, MS
365	2	Clark (Jeffersonville), IN - Scott, IN	423	2	Tangipahoa, LA - St. Helena, LA
366	4	Lorain (Lorain), OH - Erie, OH	424	5	Lafayette (Lafayette), LA - Iberia, LA
367	3	Lee, IA - Hancock, IL	425	6	Travis (Austin), TX - Williamson, TX
368	4	Fairfield (Lancaster), OH - Athens, OH	426	12	Tom Green (San Angelo), TX - McCulloch,
369	2	Richland (Mansfield), OH - Ashland, OH			TX
370	3	St. Croix, WI - Goodhue, MN	427	2	Hidalgo (McAllen), TX - Starr, TX
371	4	Alpena, MI - Presque Isle, MI	428	9	Taylor (Abilene), TX - Jones, TX
372	2	Clinton, OH - Highland, OH	429	7	Garfield (Enid), OK - Kingfisher, OK
373	3	Kane (Aurora), IL - De Kalb, IL	430	4	Cleveland (Norman), OK - Garvin, OK
374	2	Barron, WI - Rusk, WI	431	3	Warren, MS - Madison, LA
375	4	Jackson (Jackson), MI - Lenawee, MI	432	9	Pulaski (Little Rock), AR - Saline, AR
376	2	Miami (Piqua), OH - Shelby, OH	433	7	Victoria (Victoria), TX - Lavaca, TX
377	6	Muskingum, OH - Guernsey, OH	434	5	Tarrant (Fort Worth), TX - Johnson, TX
378	2	La Porte, IN - Starke, IN	435	5	Adams, MS - Concordia, LA
379	3	Shawano, WI - Langlade, WI	436	4	Grayson (Sherman), TX - Bryan, OK
380	3	Columbiana, OH - Hancock (Weirton), WV	437	9	Nueces (Corpus Christi), TX - San
381	2	Calhoun (Battle Creek), MI - Barry, MI			Patricio, TX
382	2	Racine (Racine), WI - Kenosha, WI	438	4	Lamar, TX - Red River, TX
383	2	White, IL - Hamilton, IL	439	4	Lincoln, LA - Union, LA
384	4	Saline, IL - Hardin, IL	440	2	Beckham, OK - Roger Mills, OK
385	3	Wayne, IN - Fayette, IN	441	4	Smith (Tyler), TX - Henderson, TX
386	2	Hancock, OH - Seneca, OH	442	2	Pope, AR - Yell, AR

	Number			Number	•
HSA	of	HSA name	HSA	of	HSA name
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443	7	Ouachita (Monroe), LA - Richland, LA	499	4	Crittenden (West Memphis), AR -
444	5	Ector (Odessa), TX - Ward, TX			St. Francis, AR
445	5	Muskogee, OK - Cherokee, OK	500	3	St. Tammany (Slidell), LA - Washington, LA
446	4	Washington (Fayetteville), AR - Benton,	501	2	Madison, TX - Leon, TX
447	4	AR	502	3	Grenada, MS - Montgomery, MS
447 448	4	Nacogdoches, TX - Shelby, TX Garland, AR - Hot Spring, AR	503 504	3 2	Nolan, TX - Mitchell, TX Lawrence, MS - Jefferson Davis, MS
449	5 4	Angelina, TX - Walker, TX	505	3	Brazoria (Lake Jackson), TX - Wharton, TX
450	6	Rapides (Alexandria), LA - La Salle, LA	506	7	Kerr, TX - Gillespie, TX
451	5	Washington, OK - Montgomery, KS	507	2	Erath, TX - Comanche, TX
452	4	Bell (Killeen), TX - Milam, TX	508	3	Lea, NM - Andrews, TX
453	8	Dallas (Dallas), TX - Collin, TX	509	3	Cherokee, TX - Anderson, TX
454	4	Gregg (Longview), TX - Rusk, TX	510	2	Hays (San Marcos), TX - Caldwell, TX
455	4	Jones, MS - Wayne, MS	511	2	Lincoln, MS - Copiah, MS
456	3	Harrison (Biloxi), MS - Hancock, MS	512	3	Childress, TX - Hall, TX
457	3	White, AR - Cleburne, AR	513	2	Bee, TX - Karnes, TX
458	3	Wilbarger, TX - Hardeman, TX	514	2	Terry, TX - Yoakum, TX
459	3	Lafayette, MS - Calhoun, MS	515	2	Kay (Ponca City), OK - Osage, OK
460	4	Gray, TX - Wheeler, TX	516	3	Val Verde, TX - Maverick, TX
461	4	Lowndes, MS - Clay, MS	517	1	Comal (New Braunfels), TX
462	6	McLennan (Waco), TX - Hill, TX	518	2	Howard, TX - Glasscock, TX
463	2	St. Landry, LA - Evangeline, LA	519	1	Scurry, TX
464	5	Brazos (Bryan), TX - Washington, TX	520	2	Cameron (Brownsville), TX - Willacy, TX
465	4	Titus, TX - Camp, TX	521	1	Jackson, AR
466	3	Pittsburg, OK - Pushmataha, OK	522	1	Johnson, AR
467	3	Lafourche, LA - Terrebonne (Houma), LA	523	1	Claiborne, LA
468	4	Brown, TX - Coleman, TX	524	1	Collingsworth, TX
469	5	Woodward, OK - Ellis, OK	525	1	Wilkinson, MS
470	5	Calcasieu (Lake Charles), LA -	526	3	Brewster, TX - Jeff Davis, TX
		Beauregard, LA	527	2	Arkansas, AR - Monroe, AR
471	2	Jackson (Pascagoula), MS - George, MS	528	1	St. Mary, LA
472	3	Payne, OK - Pawnee, OK	529	2	Okmulgee, OK - Okfuskee, OK
473	6	Jefferson (Pine Bluff), AR - Bradley, AR	530	1	Winn, LA
474	4	Carter, OK - Marshall, OK	531	1	Dimmit, TX
475	2	Custer, OK - Washita, OK	532	1	Galveston (Galveston), TX
476	2	Grady, OK - Caddo, OK	533	1	Yazoo, MS
477	4	Pike, MS - Marion, MS	534	1	Polk, AR
478	4	Pontotoc, OK - Seminole, OK	535	1	Beaver, OK
479	2	Palo Pinto, TX - Jack, TX	536	1	Uvalde, TX
480	2	Chicot, AR - Ashley, AR	537	1	Phillips, AR
481	3	Hale, TX - Floyd, TX	538	3	Webb (Laredo), TX - Jim Hogg, TX
482	3	Union, MS - Tippah, MS	539	5	Jasper (Joplin), MO - Newton, MO
483	3	Jackson, OK - Greer, OK	540	9	Hennepin (Minneapolis), MN - Anoka, MN
484	4	Oktibbeha, MS - Webster, MS	541	8	St. Louis, MO - St. Louis city, MO
485	3	Harrison (Marshall), TX - Cass, TX	542	9	Douglas (Omaha), NE - Dodge, NE
486	3	Union, AR - Columbia, AR	543	9	Burleigh (Bismarck), ND - McLean, ND
487	4	Leflore, MS - Bolivar, MS	544	11	Minnehaha (Sioux Falls), SD - Lake, SD
488	3	Washington, MS - Sharkey, MS	545	8	Linn (Cedar Rapids), IA - Johnson, IA
489	2	Navarro, TX - Freestone, TX	546	11	Polk (Des Moines), IA - Marion, IA
490	2	Colorado, TX - Fayette, TX	547	13	Cass (Fargo), ND - Wilkin, MN
491	2	Deaf Smith, TX - Parmer, TX	548	10	Jackson (Kansas City), MO - Clay, MO
492	5	Midland (Midland), TX - Pecos, TX	549	15	Greene (Springfield), MO - Barry, MO
493	4	Comanche (Lawton), OK - Kiowa, OK	550 551	6	Ward, ND - Bottineau, ND
494	4	Boone, AR - Carroll, AR	551	5	Ford, KS - Clark, KS
495	3	Denton (Denton), TX - Cooke, TX	552 552	8	Olmsted (Rochester), MN - Winona, MN
496	2	Ouachita, AR - Dallas, AR	553 554	7	Boone (Columbia), MO - Randolph, MO
497 498	2 2	Stephens, OK - Jefferson, OK	554 555	9	Shawnee (Topeka), KS - Riley, KS
		Alcorn, MS - Tishomingo, MS	555	5	Hall, NE - Hamilton, NE

	Number			Number	
HSA number	of counties in HSA	HSA name	HSA number	of counties in HSA	HSA name
556	6	Cerro Gordo, IA - Kossuth, IA	613	4	Red Willow, NE - Decatur, KS
557	6	Black Hawk (Waterloo), IA - Fayette, IA	614	2	Mississippi, AR - Pemiscot, MO
558	6	Pennington (Rapid City), SD - Meade, SD	615	9	Davison, SD - Charles Mix, SD
559	4	Holt, NE - Brown, NE	616	3	Phelps, NE - Furnas, NE
560	9	Woodbury (Sioux City), IA - Buena Vista, IA	617	2	Greene, IA - Guthrie, IA
561	6	Lancaster (Lincoln), NE - Gage, NE	618	3	Pratt, KS - Kiowa, KS
562	3	Baca, CO - Grant, KS	619	4	Nobles, MN - Jackson, MN
563	14	Cape Girardeau, MO - Butler, MO	621	2	Wyandotte (Kansas City), KS -
564 565	4	Adams, NE - Nuckolls, NE	600	2	Leavenworth, KS
565 566	6 6	Ellis, KS - Graham, KS	622 623	3 5	Mitchell, KS - Osborne, KS
567	4	Adair, MO - Scotland, MO Lyon, KS - Greenwood, KS	624	5	Dawes, NE - Sheridan, NE Johnson (Overland Park), KS - Douglas, KS
568	5	Codington, SD - Grant, SD	625	3	Clay, IA - Dickinson, IA
569	4	Saline, KS - Ellsworth, KS	626	2	Martin, MN - Emmet, IA
570	7	Buffalo, NE - Dawson, NE	627	4	Phelps, MO - Dent, MO
571	7	Craighead, AR - Greene, AR	628	2	Richardson, NE - Pawnee, NE
572	7	Brown, SD - Day, SD	629	6	Hughes, SD - Haakon, SD
573	4	Blue Earth, MN - Watonwan, MN	630	3	Crawford, KS - Bourbon, KS
574	8	Independence, AR - Howell, MO	631	2	Roseau, MN - Lake of the Woods, MN
575	6	Finney, KS - Scott, KS	632	3	Stutsman, ND - Foster, ND
576	6	Sedgwick (Wichita), KS - Harvey, KS	633	3	Carroll, IA - Calhoun, IA
577	5	Madison, NE - Antelope, NE	634	2	Floyd, IA - Chickasaw, IA
578	6	Yankton, SD - Bon Homme, SD	635	2	Marshall, KS - Washington, KS
579	3	Williams, ND - Divide, ND	636	2	Rawlins, KS - Cheyenne, KS
580	8	Scotts Bluff, NE - Goshen, WY	637	3	Phillips, KS - Smith, KS
581	8	Cole, MO - Callaway, MO	638	3	Pierce, ND - Rolette, ND
582	2	Otter Tail, MN - Grant, MN	639	2	Vernon, MO - Cedar, MO
583	4	Barton, KS - Rush, KS	640	6	Adams, ND - Bowman, ND
584	6	Grand Forks (Grand Forks), ND - Polk, MN	641	2	Scott (Davenport), IA - Muscatine, IA
585 586	3	Webster, IA - Humboldt, IA	642	2	Cloud, KS - Republic, KS
586 587	3	Lawrence, SD - Campbell, WY Seward, KS - Texas, OK	643 644	2 3	Greeley, KS - Wichita, KS Page, IA - Fremont, IA
587 588	4 5	Stearns (St. Cloud), MN - Morrison, MN	645	2	Lucas, IA - Fremont, IA Lucas, IA - Wayne, IA
589	6	Wapello, IA - Mahaska, IA	646	2	Freeborn, MN - Faribault, MN
590	3	Big Stone, MN - Roberts, SD	647	2	Montgomery, IA - Adams, IA
591	8	Buchanan (St. Joseph), MO - Nodaway, MO	648	5	Walworth, SD - Potter, SD
592	5	Kandiyohi, MN - Yellow Medicine, MN	649	2	Appanoose, IA - Davis, IA
593	6	Lincoln, NE - Perkins, NE	650	3	Dickinson, KS - Geary, KS
594	8	Custer, NE - Valley, NE	651	3	Platte, NE - Nance, NE
595	5	Gregory, SD - Tripp, SD	652	2	Obrien, IA - Osceola, IA
596	6	Pottawattamie (Council Bluffs), IA -	654	3	Chase, NE - Dundy, NE
		Shelby, IA	655	3	Ramsey, ND - Eddy, ND
597	2	Beltrami, MN - Clearwater, MN	656	4	Pettis, MO - Saline, MO
598	3	Reno, KS - Stafford, KS	657	2	Livingston, MO - Carroll, MO
599	3	St. Charles (St. Charles), MO - Lincoln, MO	658	2	Fillmore, NE - Thayer, NE
600	4	Beadle, SD - Hand, SD	659	3	Thomas, KS - Sherman, KS
601	4	Labette, KS - Allen, KS	660	2	Otoe, NE - Johnson, NE
602	3	Brown, MN - Redwood, MN	661	2	Harper, KS - Kingman, KS
603	3	Mille Lacs, MN - Isanti (Cambridge), MN	662	1	Clay, KS
604 605	4	Pennington, MN - Kittson, MN	663	1	Madison, MO
605 606	4	Stark, ND - Golden Valley, ND	664 665	1	Stevens, KS
607	5 3	Story, IA - Hamilton, IA Henry, MO - St. Clair, MO	666	1 3	Mitchell, IA Audrain, MO - Monroe, MO
608	3	Douglas, MN - Stevens, MN	667	3 1	Russell, KS
609	2	Lyon, MN - Lincoln, MN	668	1	Cherry, NE
610	2	Brookings, SD - Kingsbury, SD	669	1	Barber, KS
611	2	York, NE - Polk, NE	670	1	Grant, ND
612	5	Crow Wing, MN - Wadena, MN	671	1	Atchison, KS

	Number			Number	
HSA	of	. HSA name	HSA	of	. HSA name
number	counties in HSA	TISA Hame	number	counties in HSA	S H3A Hame
672	1	Sioux, IA	729	4	Malheur, OR - Washington, ID
673	1	Linn, MO	730	2	Cassia, ID - Minidoka, ID
674	1	McPherson, KS	731	6	Alamosa, CO - Rio Grande, CO
675	1	Wilson, KS	732	3	Dona Ana (Las Cruces), NM - Luna, NM
676	1	Taney, MO	733	2	Sheridan, MT - Daniels, MT
678	2	Atchison, MO - Holt, MO	734	5	Kootenai, ID - Bonner, ID
679	2	Marshall, IA - Tama, IA	735	2	Moffat, CO - Routt, CO
680	2	Dickey, ND - La Moure, ND	736	6	King (Seattle), WA - Snohomish, WA
681	1	Crawford, IA	737	4	Stanislaus (Modesto), CA - Merced, CA
682	1	Ness, KS	738	3	Coos, OR - Del Norte, CA
683 684	2 2	Grundy, MO - Mercer, MO	739 740	2	Yakima (Yakima), WA - Kittitas, WA
685	1	McIntosh, ND - Logan, ND	740 741	8	San Juan, NM - La Plata, CO
686		Camden, MO Poweshiek, IA	741 742	4 3	Laramie (Cheyenne), WY - Cheyenne, NE Silver Bow, MT - Deer Lodge, MT
687	1 1	Nemaha, NE	742	2	Klamath, OR - Lake, OR
688	11	Denver (Denver), CO - Jefferson, CO	744	5	Weber (Ogden), UT - Davis, UT
689	8	Multnomah (Portland), OR - Clackamas,	745	5	Otero, CO - Prowers, CO
000	O	OR	746	3	Solano (Vallejo), CA - Napa, CA
690	3	Yuba, CA - Sutter (Yuba City), CA	747	4	Chelan, WA - Okanogan, WA
691	8	Yellowstone (Billings), MT - Carbon, MT	748	6	Wasco, OR - Hood River, OR
692	7	Cascade (Great Falls), MT - Glacier, MT	749	2	Park, WY - Big Horn, WY
693	5	Bernalillo (Albuquerque), NM - Valencia,	750	3	San Joaquin (Stockton), CA - Amador, CA
000	Ü	NM	751	3	Santa Clara (San Jose), CA - Monterey, CA
694	6	Nez Perce, ID - Asotin, WA	752	3	Jackson (Medford), OR - Josephine, OR
695	4	Twin Falls, ID - Jerome, ID	753	2	Nevada, CA - Sierra, CA
696	5	Bannock, ID - Bingham, ID	754	5	El Paso (Colorado Springs), CO - Kit
697	3	Butte (Chico), CA - Tehama, CA			Carson, CO
698	6	Spokane (Spokane), WA - Stevens, WA	755	2	Iron, UT - Beaver, UT
699	5	Maricopa (Phoenix), AZ - Yavapai, AZ	756	2	Park, MT - Sweet Grass, MT
700	5	Pima (Tucson), AZ - Cochise, AZ	757	2	San Francisco (San Francisco), CA -
701	10	Washoe (Reno), NV- Ormsby, NV			San Mateo, CA
702	2	Benton (Kennewick), WA - Franklin, WA	758	5	Thurston (Olympia), WA - Lewis, WA
703	6	Utah (Provo), UT - Sevier, UT	759	3	Union, OR - Baker, OR
704	4	Pueblo (Pueblo), CO - Colfax, NM	760	4	Weld (Greeley), CO - Morgan, CO
705	3	Marion (Salem), OR - Yamhill, OR	761	6	Delta, CO - Montrose, CO
707	8	Clark (Las Vegas), NV - Washington, UT	763	3	Logan, CO - Phillips, CO
708	6	Salt Lake (Salt Lake City), UT - Uintah, UT	764	2	Sonoma (Santa Rosa), CA - Marin, CA
709	4	Sacramento (Sacramento), CA - Placer, CA	765	2	McKinley, NM - Apache, AZ
710	3	Shasta (Redding), CA - Modoc, CA	766	2	Alameda (Oakland), CA - Contra Costa, CA
711	6	Mesa, CO - Garfield, CO	767	4	Richland, MT - Dawson, MT
712	4	Hill, MT - Phillips, MT	768	2	San Bernardino, CA - Riverside
713	6	Missoula, MT - Ravalli, MT	760	0	(Riverside), CA
714	6	Custer, MT - Fallon, MT	769	2	Otero, NM - Lincoln, NM
715	3	Cache, UT - Franklin, ID	770 771	2	Sheridan, WY - Johnson, WY
716 717	7 4	Ada (Boise), ID - Canyon, ID	771 772	3 2	Albany, WY - Carbon, WY
717		Walla Walla, WA - Umatilla, OR		2	Chaves, NM - Eddy, NM
718	3 5	Fresno (Fresno), CA - Kings, CA Deschutes, OR - Crook, OR	773 774	2	Gallatin, MT - Madison, MT San Diego (San Diego), CA - Imperial, CA
720	3	Elko, NV - Lander, NV	775	3	Teton, WY - Lincoln, WY
720 721	3	Lewis and Clark, MT - Broadwater, MT	777	3	Fremont, WY - Hot Springs, WY
722	8	Bonneville, ID - Madison, ID	778	2	Meagher, MT - Wheatland, MT
723	2	Los Angeles (Los Angeles), CA - Orange,	779	2	Roosevelt, MT - Valley, MT
120	4	CA	780	2	Plumas, CA - Lassen, CA
724	4	Santa Fe (Santa Fe), NM - Rio Arriba, NM	781	2	Santa Barbara (Santa Barbara), CA -
725	4	Curry, NM - Quay, NM	101	_	San Luis Obispo, CA
. 20			782	2	Lane (Eugene), OR - Douglas, OR
	,	National Casper Wit-Converse Wit			
726 727	2 2	Natrona (Casper), WY - Converse, WY Linn, OR - Benton, OR	783	2	Lincoln, OR - Tillamook, OR

HSA number	Number of counties in HSA	USA nama	HSA number	Number of counties in HSA	USA nama
785	2	Clallam, WA - Jefferson, WA	807	1	Kern (Bakersfield), CA
786	2	Chaffee, CO - Lake, CO	808	2	Blaine, ID - Camas, ID
787	1	Yuma (Yuma), AZ	809	2	Carbon, UT - Emery, UT
788	1	Powell, MT	810	2	Fergus, MT - Petroleum, MT
789	1	Tulare (Visalia), CA	811	1	Mendocino, CA
790	1	Ventura (Oxnard), CA	812	2	Fremont, CO - Custer, CO
792	1	Uinta, WY	813	1	Lemhi, ID
793	3	Grant, NM - Catron, NM	814	1	Union, NM
794	1	Pierce (Tacoma), WA	815	1	Whatcom (Bellingham), WA
795	1	Boulder (Boulder), CO	816	2	Inyo, CA - Mono, CA
796	1	Larimer (Fort Collins), CO	817	3	Ketchikan Gateway, AK - Wrangell -
797	1	Platte, WY			Petersburg, AK
799	2	Sweetwater, WY - Daggett, UT	818	4	Juneau, AK - Sitka, AK
800	1	Humboldt, CA	819	3	Fairbanks North Star, AK - Southeast
801	4	San Miguel, NM - Guadalupe, NM			Fairbanks, AK
802	1	Santa Cruz (Santa Cruz), CA	820	15	Anchorage (Anchorage), AK - Kenai
803	1	Mohave, AZ			Peninsula, AK
804	1	Weston, WY	821	3	Honolulu (Honolulu), HI - Maui, HI
805	1	White Pine, NV	822	1	Kauai, HI
806	1	Grant, OR	823	1	Hawaii, HI

Traditionally, the variance for an age-adjusted death rate is computed as a weighted average of agespecific binomial variances (49). However, this estimator has been shown to often underestimate (127) and occasionally overestimate (128) the true variance of the rate. Empirical Bayes methods have been proposed to "stabilize" rates with large variances (129, 130), but these methods tend to overshrink the individual rates to some overall rate, possibly masking interesting spatial patterns (131). Mixed effects regression models were used to improve the variance estimates for the rates, and to provide predicted rates by age and region, estimates not available except from a model-based procedure. This appendix provides the technical details of the models used to produce the maps and graphs in this atlas.

Information from certificates of all deaths that occurred during 1988–92 was summarized to produce stratified numbers of deaths by cause of death (table 1), sex, race (see "Data sources" section), age (0–4, 5–14, 25–34 through 75–84, and 85 years and over), and HSA (31) for analysis. Corresponding person years at risk were computed by multiplying the stratified 1990 Census population by 5. Thus, rates presented in this atlas may be interpreted as "average annual rates" over the 5-year period.

Notation. Because separate analyses were conducted for each cause, sex, and race combination, subscripts for these factors are suppressed in this section. For each of these analyses, let

 d_{ij} = number of deaths in HSA i, age group j, n_{ij} = the corresponding population at risk.

Because deaths are rare relative to the large populations at risk, we may assume that

$$d_{ij} \sim \text{Poisson}(n_{ij}\lambda_{ij})$$
 (1)

where λ_{ij} is the age-specific death rate for age j (j=1,...,J), HSA i (i=1,...,I). Then the maximum likelihood estimator of λ_{ij} is $\hat{\lambda}_{ij} = r_{ij} = d_{ij} / n_{ij}$, the observed age-specific rate for HSA i and age j. For the atlas data, J=10 and I=798 (data for Alaska and Hawaii were not modeled).

Age-adjusted rates. The observed age-adjusted rate for HSA i is computed using the direct method as a weighted average of the age-specific rates for HSA i:

$$R_i = \sum_{j=1}^{J} r_{ij} c_j = \hat{\lambda}_i' C$$
 (2)

with expected value $E(R_i) = \lambda_i' C$, where $\lambda_i = (\lambda_{i1}, \lambda_{i2}, ..., \lambda_{iJ})'$, and C is a vector of weights consisting of age-specific proportions of the standard population $(\sum c_j = 1)$. All rates in this atlas have been adjusted to the U.S. standard million population using the age groups and weights in table 2.

Dispersion of rates. In order to make inferences about either the age-specific (r_{ij}) or age-adjusted rates (R_i) , accurate estimates of their variances are needed. In the absence of repeated data samples, the analyst needs to rely on either a computationally intensive nonparametric method, such as Gibb's sampling, or a theoretically justifiable variance estimator. A common solution is to assume homogeneity of rates across all geographic areas,

that is, $\lambda_{ij} = \lambda_j$ for all *i*. This is not a valid assumption for U.S. mortality data (131), and leads to the appearance of overdispersion of rates.

Potential sources of overdispersion in these data are heterogeneity of the underlying rates across geographic areas, exclusion of important explanatory variables from the rate model, and correlations among the age-adjusted rates. For example, rates may be spatially correlated because of unmeasured environmental, lifestyle, or other regional effects. To account for possible overdispersion, the distributional assumptions about d_{ij} (equation 1) were generalized to:

$$E(d_{ij}) = n_{ij}\lambda_{ij}$$

$$Var(d_{ii}) = \phi n_{ii}\lambda_{ii}$$
(3)

Then asymptotically, $\operatorname{Var}(r_{ij}) = \phi \lambda_{ij} / n_{ij}$ and

$$\operatorname{Var}(R_{i}) = \phi \sum_{j=1}^{J} c_{j}^{2} \frac{\hat{\lambda}_{ij}}{n_{ij}} = \phi C' \operatorname{Diag}\left(\frac{\hat{\lambda}_{ij}}{n_{ij}}\right) C \qquad (4a)$$

Because death is a rare event, this Poisson-derived variance is well approximated by the binomial form of the variance:

$$\operatorname{Var}(R_i) \approx \phi C' \operatorname{Diag}\left(\frac{\hat{\lambda}_{ij}(1 - \hat{\lambda}_{ij})}{n_{ij}}\right) C$$
 (4b)

which is just the traditional variance for an ageadjusted rate (49) multiplied by the dispersion factor ϕ . **Generalized linear model.** In order to share information across areas to achieve stability of parameter estimates, the place- and age-specific rates were modeled as a function of age. That is,

$$\ln(r_{ij}) = f(a_j, \beta_i) + \varepsilon_{ij}$$
 (5)

where a_j is the midpoint of age group j rescaled by dividing by 10 (that is, $a_j = 0.25, 1, 2,..., 9$), and β_i is the vector of parameters to be estimated; 0.000001 was added to each r_{ij} to avoid taking logarithms of zero. Because of the irregularity of the dependence of death rates on age for external causes of death, it was necessary to use cubic and linear spline forms for $f(a_j,\beta_i)$. Specifically,

$$f(a_j, \beta_i) = \ln \lambda_{ij} = X_j \beta_i \tag{6}$$

For the linear model

$$X_{j} = (1, a_{j}, \delta_{1}(a_{j} - k_{1}), \delta_{2}(a_{j} - k_{2}), \delta_{3}(a_{j} - k_{3}))$$
 (7a)

and for the cubic model

$$X_{j} = \left(1, a_{j}, a_{j}^{2}, a_{j}^{3}, \delta_{1}\left(a_{j} - k_{1}\right)^{3}\right)$$
 (7b)

where $\delta_{\scriptscriptstyle m}=1$ if $a_{\scriptscriptstyle j}>k_{\scriptscriptstyle m}$, 0 otherwise, and $\{k_{\scriptscriptstyle J},\,k_{\scriptscriptstyle 2},\,k_{\scriptscriptstyle 3}\}$ is the set of spline knots predetermined as optimal for U.S. age-specific rates for each cause, race, and sex. Thus this model reduced the number of parameters to be estimated from 10 (λ_i) to 5 (β_i) for each HSA.

Random effects. Further reduction in the parameter space and stability of estimates were achieved by allowing the intercept and, for whites, initial slope parameter estimates to vary by HSA within region in a hierarchical random effects model. That is,

$$\beta_{i} = \beta_{k} + b_{i} \text{ for HSA } i \in \text{region } k$$

$$b_{i} = \begin{cases} (b_{0i}, b_{1i}, 0, 0, 0)' & \text{for whites} \\ (b_{0i}, 0, 0, 0, 0)' & \text{for blacks} \end{cases}$$

$$\beta_{k} \text{ fixed and } b_{i} \sim \text{N}(0, D_{k})$$
(8)

where D_k is the covariance matrix for the random HSA effects. Because of computational constraints, all

regional effects were considered fixed, not themselves random effects within an overall U.S. effect. Predicted age-specific rates derived from these regional estimates, $e^{X_j^c \hat{\beta}_k}$, are plotted along with their 95-percent confidence limits for each cause, race, and sex (see figure 1c). This model accommodates age-by-HSA interactions through the HSA random effects within region and age-by-region interactions through the individual fixed regional effects.

Computational methods. From equation 4a, it can be shown that asymptotically

$$\operatorname{Var}\left(\ln\left(r_{ij}\right)\right) \approx \phi/\left(n_{ij}\lambda_{ij}\right) \tag{9}$$

Therefore, a generalized linear model analysis of $\ln(r_{ij})$ using inverse standard error weights of $\left(n_{ij}\lambda_{ij}\right)^{1/2}$ will yield an estimate of the dispersion parameter ϕ directly as the residual variance:

$$\hat{\phi} = \sum_{i,j} \left[\ln(r_{ij}) - E(\ln(r_{ij}) | \beta_i) \right]^2 / (IJ - p)$$
 (10)

where p is the number of parameters estimated for the model.

An EM algorithm can be used to estimate the parameters, by iterating between computing the weights $\left(n_{ij}\lambda_{ij}\right)^{1/2} = \left[\mathrm{E}\!\left(d_{ij}\big|\lambda_{ij}\right)\right]^{1/2}$ from each $\hat{\beta}_i$, and then recomputing $\hat{\beta}_i$ using these new weights. Initial weights were created by substituting d_{ij} for $\mathrm{E}\!\left(d_{ij}\big|\lambda_{ij}\right)$ unless $d_{ij} < 3$, when an expected number of deaths was computed using marginal regional rates. Parameters were estimated by repeated applications of SAS PROC MIXED (132).

Analysis of sample data demonstrated convergence of the parameter estimates within five iterations. In fact, except for very sparsely populated areas, the results of the first PROC MIXED model using the initial weights were within 5 percent of the final rate estimates. Therefore, because of the computational difficulty of iterating the random effects model for each of the 72 cause-, race-, sexspecific datasets, the results of the initial iteration were used as input to the smoothing algorithm.

Smoothing algorithm. Predicted age-specific rates for each HSA were smoothed using a weighted headbanging algorithm (52), with weights equal to the inverse of the rates' estimated standard errors. For the sample data, this choice of weights caused rates for sparsely populated HSA's to be smoothed to

essentially the same relative values whether results of the first or last iteration were used. These smoothed, modeled age-specific rates were then mapped for each cause, race, and sex as an adjunct to the maps of age-adjusted rates (see figures 1d, 1e).

Variance estimation. An analysis of simulated data showed less than a 1-percent bias in estimating $\hat{\beta}_i$ using these methods, but a large bias in estimating ϕ . Therefore, a robust sandwich estimator was used to compute the dispersion parameter (133):

$$\hat{\phi} = \sum_{i,j} \frac{\left[d_{ij} - \mathrm{E}(d_{ij}|\beta_i)\right]^2}{\mathrm{E}(d_{ij}|\beta_i)} / (IJ - p)$$
(11)

Observations with absolute standardized residuals more extreme than 5 were excluded from equation 11. This estimator was also proposed by McCullagh and Nelder (134) as a residual sum of squares estimator of dispersion.

Several forms of the random effects variance matrix D_k were examined using PROC MIXED, several of which included spatial correlation among HSA's as a function of distance between population centroids. However, after accounting for broad spatial patterns through the fixed regional effects, the covariance parameters converged to zero. Similarly, examination of the residuals showed little remaining spatial autocorrelation. Thus the final models used for the atlas data included only diagonal variance matrices.

The robust dispersion estimate (equation 11) was used to adjust the traditional variance for each age-adjusted rate (as in equation 4b) for purposes of determining reliability (figure 1a) and significance (figure 1b) of the rates for mapping. When applied to simulated data, this method resulted in 94.5-percent coverage of the true rates by nominal 95-percent confidence intervals. Goodness-of-fit plots and statistics indicate that these methods fit the observed mortality data well (48).

In response to requests from public health researchers who have used other statistical rate atlases, a number of supplemental maps have been included in this appendix to aid in interpreting the mortality maps.

Population maps. Figure 5 presents the agerace-modified 1990 population counts that were multiplied by 5 to provide the rate denominators. Although very broadly categorized, these maps illustrate the patterns of population distribution throughout the United States and can be used to identify HSA's where the age-adjusted rates are based on small populations. Figures 6 and 7 provide the percent of the sex- and race-specific populations in each HSA that fall into age groups 15–24 years, 35–44 years, and 65–74 years. Ages 20 years and 70 years were used for the smoothed maps (figures 1d, 1e) for external causes of death and ages 40 and 70 years were used for the remaining causes.

Correlate maps. Figures 8 and 9 include maps of several demographic and lifestyle factors that may be associated with several of the causes of death in this atlas. These maps may be helpful in formulating etiologic hypotheses for the causes of death included in this atlas, although no claims are made as to the validity or appropriateness of these particular estimates for drawing inferences about such links.

Figure 8 includes mapped indicators of income and education by county for the total population. Percent of the total population of Hispanic origin is also shown in figure 8 to identify areas with a potentially large contribution of Hispanic patterns of mortality to the rates mapped for whites.

Figure 9 presents State maps of selected modifiable lifestyle factors among adults (ages greater than or equal to 18) as estimated by the Behavioral Risk Factor Surveillance System in 1991 (135). These factors are briefly defined:

- Smokers—Current regular use of cigarettes by someone who has ever smoked at least 100 cigarettes.
- Overweight adults—Body mass index of at least 27.8 for men and 27.3 for women.
- Seat belt users—Seat belt worn whenever driving or riding in a car.
- Sedentary lifestyle—No exercise, recreation, or physical activities other than regular job duties during the previous month.

FIGURE 5. POPULATION BY HSA, 1990

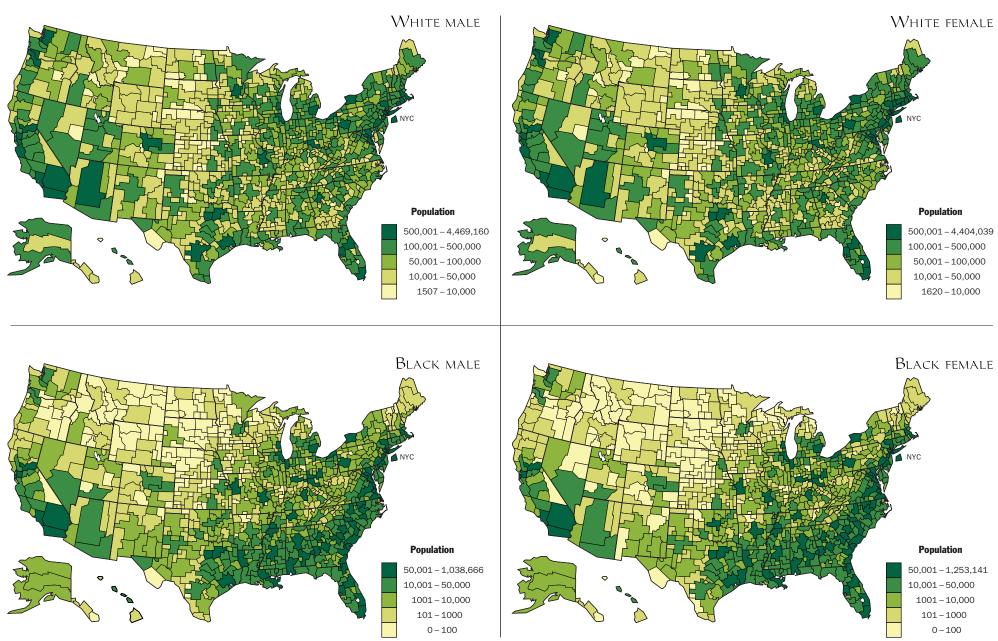
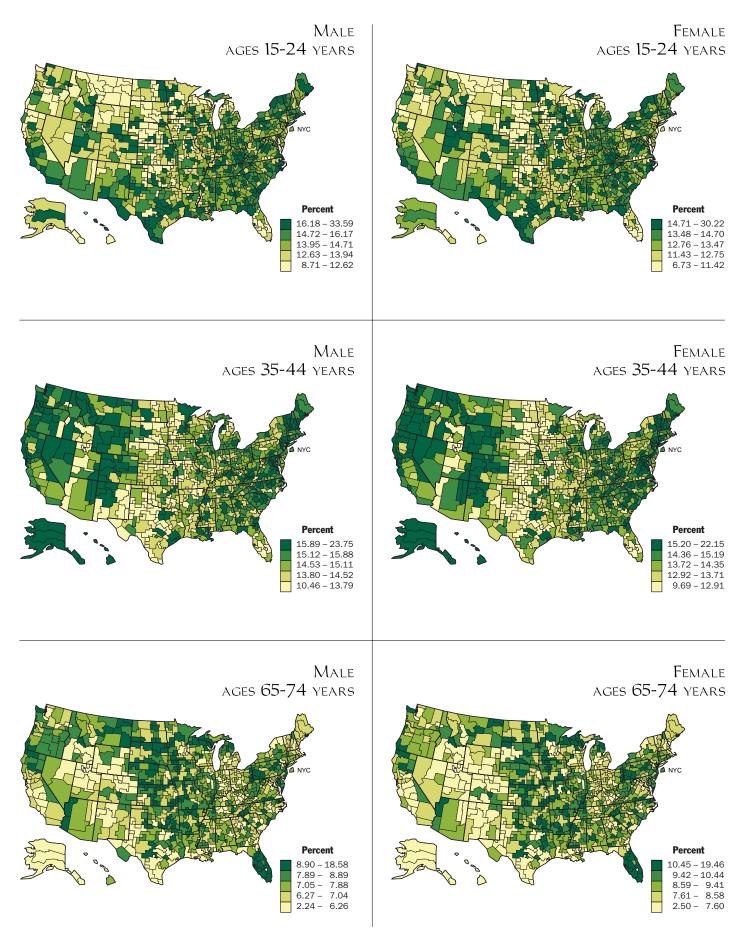
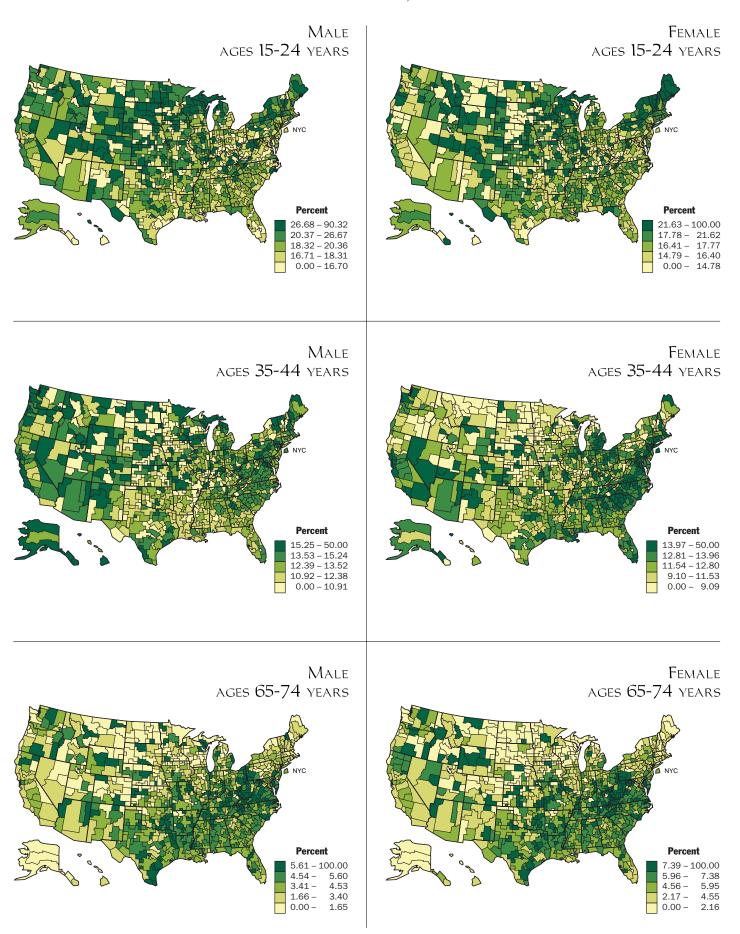


FIGURE 6. PERCENT OF TOTAL HSA WHITE POPULATION IN EACH REPRESENTATIVE AGE GROUP, 1988-92



 ${\tt SOURCE: U.S. \ Bureau \ of the \ Census \ (age/race/modified \ census \ counts)}.$

FIGURE 7. PERCENT OF TOTAL HSA BLACK POPULATION IN EACH REPRESENTATIVE AGE GROUP, 1988-92



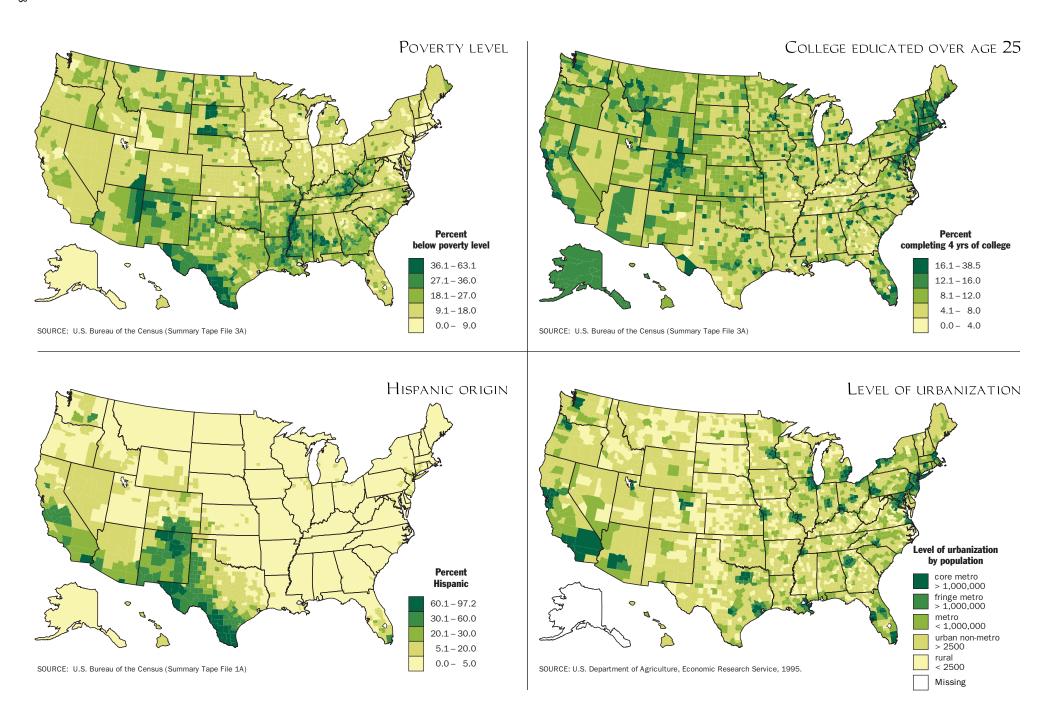


FIGURE 9. CORRELATE VARIABLES BY STATE, 1991

